Haematinic activity of

PULI ILAI CHOORANAM

(Tamarindus indica (L))

&

Hepatoprotective activity of

Chara Parpam

Dissertation submitted to

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Arumbakkam – 600 106.

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GOVT. SIDDHA MEDICAL COLLEGE, CHENNAI-106

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"Haematinic activity of *Puli ilai chooranam* (*Tamarindus indica* (L)) and Hepatoprotective activity of *Chara Parpam.*"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.V.Velpandian,M.D (S), Ph.D.,** Post Graduate Department of *Gunapadam*, Govt.Siddha Medical College, Arumbakkam, Chennai-106 and the dissertation has not formed the basis for the award of any Degree, Diploma, Fellowship or other similar title.

Date: **Signature of the Candidate**

Place: Chennai **P.Arunmozhi**

GOVT. SIDDHA MEDICAL COLLEGE, CHENNAI-106

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled "**Haematinic activity of Puli ilai chooranam (*Tamarindus indica* (L)) and Hepatoprotective activity of *Chara Parpam*"** is submitted to the Tamilnadu Dr.M.G.R.Medical University in partial fulfillment of the requirements for the award of degree of M.D (Siddha) is the bonafide and genuine research work done by P.Arunmozhi Under my supervision and guidance and the dissertation has not formed the basis for the award of any Degree, Diploma, Associateship, Fellowship or other similar title.

Date: **Seal & Signature of the Guide**

Place: Chennai **Dr.V.Velpandian,M.D (S), Ph.D.,**

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **“**“**Haematinic activity of Puli ilai chooranam (*Tamarindus indica* (L)) and Hepatoprotective activity of *Chara Parpam*** is a bonafide work carried out by **P.Arunmozhi** under the guidance of **Dr.V.Velpandian,M.D (S),.Ph.D.,** Post Graduate Department of Gunapadam, Govt.Siddha Medical College, Chennai - 106.

Seal & Signature of the HOD **Seal &Signature of the Principal**

Date: **Date:**

Place: Chennai **Place:** Chennai

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This dissertation is dedicated to my father **K. Poiyamozhi** and to all my Family.

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ABBREVATIONS:

Ab	-Accessory bundle
AbS	-Abaxial side;
Ads	– Adaxial side;
Alb	– Albumin
Alb	-Albumin
ALP	- Alkaline phosphatase
ALT	- Alanine transaminase
ANOVA	-Analysis of variance
AST	- Aspartate transaminase
AT	- After treatment
BT	- Before treatment
CCL_4	- Carbone tetrachloride
Co	-Collenchyma
CPCSEA	- Committee for the purpose of control and supervision of experimental animals.
Cu	-Cuticle
Dc	– Differential Count
Dep	- Deposits
E	– Eosinophil
EDG	- Esophagogastroduodenoscopy
Ep	-Epidermis
ESR	– Erythrocyte Sedimentation Rate

FEC	– Few Epithelial Cells
FPC	– Few Pus Cells
FPC	-Few Pus Cells
Hb	– Haemoglobin
IEC	- Institution ethical committee.
L	– Lymphocyte
L	- Lymphocyte
Lep	-Lower (Abaxial) epidermis
LFT	- Liver function test
LM	– Leaf Margin;
MAPA	- Medicinal and Aromatic Plants Abstracts
MVB	– Median Vascular Bundle;
OEC	– Occasional Epithelial Cells
OPC	– Occasional Pus Cells
P	– Polymorph
P	- Polymorphs
P	-Parenchyma
Pa	- Palisade tissue
PCS	- Pus Cells Green
Pf	-Pericyclic fibre
Ph	– Phloem;
Pi	-Pith
PUD	- Peptic ulcer disease
RFT	- Renal function test

SBS	– Sclerenchymatous Bundle sheat;
Sf	-Sclerenchyma fibre
Sp	-Spongy tissue
St	-Stoma
Sug	– Sugar
TC	– Total Count
TC	- Total count
TLC	- Thin layer Chromatography
Uep	-Upper (Adaxial) epidermis
Vb	-Vascular bundle
Vc	-Vascular cylinder
Vi	-Vein islet
Xy	-Xylem

1. INTRODUCTION

Tamil medicine, being the ancient system in the world has a stringer attraction towards it because of its detoxification, Anti – oxidation, Immune modulation and metabolic balance. Since it has given away many incredible and rapid results in various unremitting ailments, the system has been carefully guarded. Without the aid of microscope, our ancient saints – Siddhars were able to identify innumerable herbs, their properties, purification process, also administered plant based and mineral based medicine with suitable adjuvant and they documented all these things in the form of manuscripts and handed over to their disciples over generation. The therapeutic effect of the system has been proved for thousands of years that were never changed for even day to day practice.

Since, Siddha medicine is an individualistic medicine, on patients constitution and syndrome differentiation we cannot make use of modern medical system as a standard to explain in all levels. Our Siddha medicine not only pays attention to the preventive and curative methods, but also focussed on the physical, mental, spiritual and psychological well-being thus giving a total perfection in life.

Recently people diverted their attention towards eco – friendly & bio – degradable organic plant based products for the prevention of many threatening diseases. It is documented by WHO that 80% of the world's population has faith in traditional medicine, particularly plant drugs for their primary healthcare. (Dubey, et al 2004)

Anaemia is a global public health problem affecting both developing and developed countries with major consequences for human health as well as social and economic development. It occurs at all stages of the life cycle, but is more prevalent in pregnant women and young children. In 2012, iron deficiency anaemia (IDA) was considered to be a curse and deadlock for the development of the nation. Although the primary cause is iron deficiency, it is seldom present in isolation. More frequently it coexists with a number of other causes, such as malaria, parasitic infection, nutritional deficiencies, and Haemoglobinopathies. Several plants are used in treating anaemia and many are used effectively for their Haematinic properties in Siddha medicine.

I selected "*PANDU NOI"* which can be correlated with the clinical condition called iron deficiency Anaemia for my dissertation work. *Pandu Noi* is a very common disease in humans due to malnutrition, ignorance, poverty and poor socio –economic condition and also among the affluent and well fed due to unbalanced diet. In spite of the sophisticated advancement, the modern system of medicine does not provide a permanent solution to many chronic aliments. This work is done with an aim to come out with satisfactory results for one such chronic ailment named *PANDU NOI* (Anaemia).

Basically I am interested in the well being of all human being irrespective of their economic status. I have witnessed many patients in the outpatient department during my graduation, who are the victims of Anaemia and most of them are below the poverty line. This kindled a spirit in me to carry out a work in Anaemia and come out with a most effective medicine at low cost effect. Healthy India reflects a developed India. So in order to achieve the goal of healthy India this work seems to be a limelight to some extent. This is the main motivation for the work done.

Pandu noi is one of the conditions which has to be treated promptly with utmost care and requires a pragmatic approach. Any failure to treat *Pandu noi* can lead to dangerous end often fatal consequences. So I selected *Puli ilai Chooranam* to study its Haematinic Activity.

2. AIM AND OBJECTIVES

AIM

Anaemia is a major killer in India. Statistics reveal that every second Indian woman is anaemic and one in every five maternal deaths is directly due to anaemia. Anaemia spares none; it affects both adults and children of both sexes, although pregnant women and adolescent girls are most susceptible and most affected by this disease.

Through the ages, man had learnt to utilize the natural resources placed at his dispoal, to satisfy his essential needs in all field. As important reserves and sources of abundance, natural resources are indispensable for socio – economic development. According to the literature of siddha the *Puli ilai* (*Tamarindus indica*) helps to cure *Pandu noi* (Anaemia). The principle aim of this study is to evaluate the efficacy of the *Puli ilai chooranam* in the management of *Pandu noi* (Anaemia) in Pre – clinical and Clinical aspect.

OBJECTIVE

The following machinery was carried out in this dissertation.

- To study the Pharmacognostic features of the plant *Puli ilai (Tamarindus indica)* this includes the taxonomic identification of the plant with Macro, Microscopical information.
- To collect the relevant literature from classical Siddha literature as well as Modern sciences that supports this study.
- To prepare the drug according to Siddha literature.
- Physio – Chemical and Phyto – Chemical analysis for the trial drug to identify the active components.
- To study the acute toxicity of *Puli ilai chooranam* according to OECD guidelines 425.
- To establish the pharmacological activity of *Puli ilai chooranam.*
- To review the therapeutic potential of the drug through clinical trail for the management of *Pandu noi (Anaemia).*
- To scrutinize all the above study results to evaluate the efficacy of *Puli ilai Chooranam* in patients with anemia.

3. REVIEW OF LITERATURE

3.1. Botanical aspects

Botanical name : *Tamarindus indica (L)*

Family name : Caesalpinaceae

Scientific classification:

Kingdom	:	Plantae - Plants
Subkingdom	:	Tracheobionta -Vascular plants
Superdivision	:	Spermatophyta - Seed plants
Division	:	Magnoliophyta - Flowering plants
Class	:	Magnoliopsida - Dicotyledons
Subclass	:	Rosidae
Order	:	Fabales
Family	:	Caesalpinaceae
Genus	:	Tamarindus L. - Tamarind
Species	:	*Tamarindus indica L. - Tamarind*

Vernacular names:

Sanskrit	: *Tintiri, Tintrani, Amlika, Tintidr, Tintili, Ambia*
English	: Tamarind tree
French	: *Tamarinier*
Germany	: *Tamarindi*
Punjab	: *Imli*
Telugu	: *Chinta – Pandu, Asek*
Tamil	: *Puliyan, Puli.*

PLANT DESCRIPTION:

Figure.1. Showing the leaves of *Tamarindus indica (L.).*

Tamarind is a slow-growing enormous tree that can reach a height of 80 or even 100 feet (24-30 m), spread a crown of 40 feet (12 m) and develop a very large trunk of 25 feet (7.5 m) in circumference. It is a long-lived tree with high resistance to wind dark-gray and rough bark and strong, supple branches that are gracefully drooping at the ends. The mass of bright-green, fine, feathery foliage is composed of pinnate leaves, each having 10 to 20 pairs of oblong leaflets, which fold at night. The leaves are normally evergreen but may shed briefly in very dry areas during the hot season. Inconspicuous, inch-wide flowers, born in small racemes, are 5-petalled (2 reduced to bristles), yellow with orange or red streaks. The flower buds are distinctly pink due to the outer colour of the 4 sepals, which are shed when the flower opens. The fruits are curved and bulged pods, borne in great abundance along the new branches. The pods are cinnamon-brown or greyish-brown and tender-skinned with green, highly acid flesh and soft, whitish and under-developed seeds. As they mature, the pods fill out and the juicy, acidulous pulp turns brown or reddish-brown. Then, the skin becomes a brittle, easily cracked shell and the pulp dehydrates naturally to a sticky paste enclosed by a few coarse strands of fibre.

Habitat:

This evergreen tree which is indigenous to South India and it is cultivated throughout India and Burma.

Part used:

Pulp of the fruit: Seeds, Leaves, Flowers and Bark

Chemical Constituents:

Leaves: Moisture, Protein, Fat, Fibre, Carbohydrates ,Minerals.

The mineral and vitamin constituents were as follows;

Calcium, Magnesium, Phosphorus, Iron, Copper, Chloride, Sulphur, Thiamine, Riboflavin, Niacin, vitamin C.

Pulp contains Tartaric Acid. Citric acid, Malic acid and Acetic acids, Tartaric of Potassium. Invertsugar, Gum and Pectin, Seeds testa contains a Fixed oil and insoluble matter. Seeds contain Albuminoids, Fat, and Carbohydrates. Fibre and Ash containing Phosphorus and Fibrinogen Fruit contains trace of oxalic acid.

Action:

Unripe fruit is highly acid, pulp of ripe fruit, which is sweet or acid, is cooling (Refrigerant), Carminative, digestive and laxative: a valuable Antibilious. Leaves and flowers are cooling and Antibilious. Red outer covering of seeds is a mild astringent. Bark is astringent and tonic.

Uses:

Leaves crushed with water and expressed yield an acid fluid useful in bilious fever and in the scalding of the urine. Leaves crushed with water and made into a poultice are applied in inflammation of ankle joints, etc. to reduce swelling to relieve pain. Thick syrup of pulp and leaves boiled will heal up swellings with great heat and burnings.

3.2. GUNAPADAM ASPECT

புளியிலை:

பொதுகுணம்:

அழுபுண்ணை நீக்கும் அடல்சோபை மாற்றும்
எழுபாண்டு வைப்போக்கும் இப்பால் - முழுதும்
அளியச் சிவந்தகண்ணோ யாற்றுங் கனலாம்
புளியிலையை நன்றாய்ப் புகல். - அகத்தியர் குணவாகடம்

இது அழுகியபுண், சோகை, பாண்டு, சிவந்த, கண்ணோய், இவைகளை நீக்கும்.

பயன்கள்:

இலை ஒரு பங்கு, வேப்பிலை ஒரு பங்கு இவ்விரண்டையும் சேர்த்திடித்து 8 பங்கு நீர்விட்டு காய்ச்சி புண்களை கழுவி வர ஆறாதபுண்கள் ஆறிப்போகும்.

இலைச்சாற்றில் பழுக்கக்காய்ந்த இருப்பு சலாகையைத் தோய்த்து அச்சாற்றில் ஒன்றிரண்டு சங்குவீதமெடுத்துச் சீதகழிச்சலுக்குக் கொடுக்கலாம்.

புளியிலை சேரும் மருந்துகள்:

1. **பாண்டு முதலானவைகளுக்கு - சிஞ்சாதி இலேகியம்.**

புளியிலை 3500கிராம், துருப்பிடித்த இரும்பு 1050கிராம் எடுத்து. தண்ணீர்26 லிட்டர் விட்டு அடுப்பின் மேலேற்றி 2லிட்டர் குடிநீர் ஆகும்வரை சுண்டக்காய்ச்சவும்.

பழைய வெல்லம் 1750கிராம் குடிநீருடனேகரைத்து மேற்புறணி நீக்கிய இஞ்சி 560 கிராம் கூடப்போட்டு மறுபடியும் அடுப்பின் மேலேற்றிப் பாகாக்கிக் கொள்ளவும்.

சீரகம்
கருஞ்சீரகம்
சுக்கு
மிளகு
திப்பிலி

இவைகளை வகைக்கு 70கிராம் எடுத்து இடித்து வத்திரகாயம் செய்துகொண்டு
சுத்தித்த இரும்புத்தூள் - 35கிராம்
சுத்தித்த மண்டூரத்தூள் - 35கிராம் கலந்து

பாகில் போட்டுக்கிளறவும். இலேகிய பதத்திலிறக்கி ஆறியபின் சுத்தமான தேன்500மி.லி விட்டு குழப்பிக்கொள்ளவும்.

இந்த இலேகியத்தை தினம் இருவேளை கொடுக்கப் பாண்டு. காமாலை, முதலிய இரோகங்கள் தீரும்.

பத்தியம்.

புளி, உப்பு, கடுகு, நல்லெண்ணெய், வாயுபதார்த்தங்கள், இறைச்சி, முதலியவைகளை நீக்கவும்.

குறிப்பு.

இரும்பைச்சுத்தி செய்யும்முறை முதல்நாள் நல்லெண்ணெயில் ஊறவைத்து மறுநாள் வறுக்கவும்.

மண்டூரம் பசுமூத்திரத்தில் ஊறவைத்து கழுவிவிடவும்

2. பாண்டு முதலானவைகளுக்கு அயபற்பம்.

இரும்புத்தூளை ஒருநாள் முழுவதும் நல்லெண்ணெயிலூற வைத்து மறுநாளதை அடுப்பில் மேலேற்றி வறுத்துக்கொள்ளவும். இதையொரு கல்வத்திலிட்டு

நெல்லிப்பருப்புக் கசாயம்

புளி இலைச்சாறு

நாகப்பட்டைச்சாறு

இவைகள் விட்டுத்தனியே நன்றாக அரைத்து வில்லைகள் தட்டி அகலில் வைத்துச்சீலைமண் செய்து. கசபுடம் (1000 வறட்டி) இடவும். இவ்வாறு 5 புடங்கள் போடவும். சுத்தமான பற்பம் கிடைக்கும்.

தக்க அனுபானங்களில் சாப்பிடச் **பாண்டு, சோகை, காமாலை,** முதலான நோய்கள் தீரும்.

தேன் (அனுபான மருந்து):

செய்கைகள்: (குணபாடம்- தாது,சீவ வகுப்பு)

உள்ளழலாற்றி மலமிளக்கி

துவர்ப்பி அழுகலகற்றி

கோழையகற்றி போஷணகாரி

பசித்தீத்தூண்டி தூக்கமுண்டாக்கி

சுத்தி:

- நீர் எந்திரத்திலிட்டுக் காய்ச்சி சூடாயிருக்கும்போதே, ஈரக்கம்பளியிலிட்டு வடிகட்டிக் கொள்ளவேண்டும்.
- ஓட்டைச் சுட்டுத் தேனில் போட்டு உபயோகிப்பது நாட்டு வழக்கம்.

குணம்:

“ ஐயயிரும லீளைவிக்க லக்கிப்புண் வெப்புடல்நோய்
பைய வொழியும் பசியுமுறுமம் - வையகத்தி
லெண்ணுமிசை யாமருந்திற் கேற்ற வனுபான
நண்ணுமலைத் தேனொன்றி னால்”.

உபயோகங்கள்: தேன் ஒரு சிறந்த துணை மருந்தாகும்

அனுபானப் பொருளாவதன்றி அவிழ்தப் பொருளாகவும் இருந்து தேகத்தை நன்னிலையில் வைத்து, வாத முதலிய முக்குற்றங்களையும் போக்குகின்றது.

“அனுபான மாய்ப்பின் அவிழ்தமுமாய்த் தோன்றி
கனமான தேகநிலை காட்டிப்- பினுமே
யரசன் முதல்வோ ரையுமாட்டு வித்தாலே
பிரசத் தினாற்போம் பிணி”.

– தேரன் பொருட்பண்பு நூல்.

அவிழ்தம் பலிக்க வேண்டுமாயின் அனுபானப்பொருள் தேவை என்பதையும், அவ்வனுபானப் பொருட்களில் தேனும் ஒன்று என்பதனையும்,

குழந்தைகளின் இருமலுக்குத் தேன் 60 மி.லி, எலுமிச்சை பழரசம் சமஅளவு கூட்டி கொடுக்க தணியும்.

- தேனைப் பானகம் செய்து வந்தால் கபப்பிணிகள் தீரும்,

  “இறவுளர் அமுதையை இறவுளதாக்கும்” என்ற தேரன் கரிசல் அடியால் அறியலாம்.
- தேனை சூடுள்ள பார்லி அரிசி கஞ்சியுடன் கொடுக்க மலபந்தம், செரியாமை,
- நீர்க்கோவை, இருமல், தொண்டை விரணம், நுரையீரல் சம்மந்தமான பிணிகள் தீரும்.

3.3. SIDDHA ASPECT OF THE DISEASE

Synonyms: ***Vemmai noi, Pandu noi.***

Nature of the disease:

The natural colour of the body will be found changed, the body will appear pale. On examination of conjunctiva and nails, they appear pale due to loss of blood.

Genesis of the disease:

It is considered that the disease may be caused by the following factors:

By eating diets of too much salty and sour which will impair the potency of blood, as a sequelae to fever, diarrhoea, vomiting and arthritis, as a sequelae to diseases such as polymenorrhoea, blood heat disease (*kuruthiazhal),*bloody diarrhea, haematemesis and haemorroids which cause loss of blood from the body, by taking drugs which are too toxic in excessive doses, worm infestation, fatigue, diarrhea with mucus, liver diseases which will impair haemopoiesis, frequent ingestion of tobacco, betel leaves and arecanut, sand, ashes, sacred ashes, and camphor.

Prodromal symptoms:

The *Pitha* dosha becomes excessive in activity due to factors such as diet and impairs the colour and volume of the blood. In addition, it also makes the body pale without affecting the nutritional requirement of the body. Later patient may develop fatigue of legs even walking for a short distance, dyspnoea, anorexia, nausea, giddiness, diminution of vision, frequent fainting, palpitation and emaciation of the body.

Types of disease:

The disease has been classified into five types. Of these, four types are developed due to dosha and one type due to toxicity. They are:

1. *Vali Pandu* (Anaemia due to derangement of *Vatha* humour)
2. *Pitha Pandu* (Anaemia due to derangement of *Pitha* humour)
3. *Kapha Pandu* (Anaemia due to derangement of *Kapam* humour)
4. *Mukutra Pandu* (Anaemia due to derangement of *Mukktra* humour)
5. *Nanju Pandu*. (Due to toxicity)

Besides, some ancient siddhars classified into one more type, viz. Anaemia due to sand eating. In addition, due to deficiency of blood the body will be emaciated and odematous: the colour of the body will be changed and appear as yellow or blue; the patient will develop excessive thirst, frequent giddiness and mental depression and impairment of learning. Further, in men diminished sexual potency may also occur. Some ancient physicians have classified this type of the disease into three sub – types as *Neela paandu*, *Alasa paandu* and *Alimuga paandu*; however, all these sub types have the clinical features of anaemia (*veluppu noi*), it is considered that there is no need to mention them separately.

Signs and Symptoms

Vali Pandu;

In this disease, there will be abdominal pain. Patient will have anorexia and thirst. In addition, the blood vessels will appear black and tortuous. There will be also shivering of the body with congestion of the eyes; the body will also appear pale and oedematous; there may be pain at the oedematous sites. The bowels will be constipated and the stool will be hard. Ageusia, flatulence and colic pain are other features of the disease.

Pitha Pandu:

In this disease, body will become yellow in colour. The tongue, the upper and lower limbs will become pale: there will be diminution of vision. In addition, patient will

have excessive thirst and bitter taste in the mouth; patient may also develop giddiness; sometimes patient may also develop bitter taste. The other features of the diseases are; breathlessness as if the chest is constricted and giddiness; these features are called as *Pitha pandu.*

In addition to the above mentioned clinical features, the following signs and symptoms may also appear; desire to eat cold diet, flatulence bad odour in the mouth, paleness of the body with yellow discolouration, ulcers in the mouth and frequent diarrhoea with stool appearing yellow in colour.

Kapha Pandu:

The skin will appear pale and the veins will become prominent, salty taste in the tongue, rising of the hairs of the body, vomiting and hoarseness of voice, frequent sneezing, expectoration of sputum, fainting attacks, pain in the hip, diminished sexual potency in male, getting angry frequently, fatigue and tiredness.

Mukktra Pandu:

Wheezing, dyspnoea, fatigue, loss of body strength with palpitation in the chest, body becoming hot and frequent micturation, body becoming hot with fainting, sneezing, frequent occurrence of oedema of the whole body.

Nanju Pandu:

In this disease, the body will become pale due to toxicity of the foods taken which are allergic. Excessive thirst, ageusia, vomiting, hiccup, cough, breathlessness and generalized anasarca are other features of the disease. In addition, there may be venous engorgement with body becoming hot.

***Pandu* due to sand eating;**

The disease usually occurs in infant and young children and also in pregnant women who develop desire to eat sand, ash, brick, sacred ashes and camphor. The anaemia develops due to excessive intake of these substances.

In this disease, depending upon the nature of the substances taken. Abdominal distension, indigestion, vomiting, diarrhoea will develop. In addition, worm infestation may also occur and the body will become lean. The blood volume also will decrease and body will become pale and oedematous. Patient may also develop palpitation.

Curable and incurable disease;

Above type of anaemia's where uncontrolled vomiting or diarrhoea, excessive oedema of the body, excessive thirst, hiccup and cough develop are considered as disease which cannot be easily cured: in addition toxic anaemia is also not easily curable disease.

General features;

There will reduction of body strength day by day and patient may not be in a a position to walk , in addition the following features may also appear ; headache, palpitation, frequent blurring of vision , vertigo, giddiness, breathlessness, anorexia, dislike to food and vomiting even if small quantity of food ingested, body becoming pale with shrinkage of skin, body become lean with pale and shiny appearance, clubbing of nails ,ulcer of the tongue or red appearance of the tongue as if the outer layer of the tongue has been scrapped ; sometimes, the tongue may also be pale with lubricity appearing like silk cloth and sore throat.

If the disease occurs in women, the menstrual blood flow will be scanty and discoloured. However, in some women, there may be excessive menstrual blood flow. The disease may also occur as an associate illness of worm infestation and blood pitha disease which occur in children and elderly.

For those people whose body is hot by nature the disease appears with the following clinical features; anaemia occurs first, indigestion of food taken, burning sensation of the body and sensation of fever, the tongue may become pale and appear red or it may become lubricant like silk cloth, inability to chew or swallow the food, frequent vomiting with small amount of bile, bitter taste of mouth, stomach disease and abdominal pain with frothy diarrhoea.

If the signs and symptoms mentioned above increase day by day, the blood volume will decrease and will be discoloured. In addition the body will appear yellow as if jaundice has occurred. When the disease advances the patient may also develop the following features.

Dosha and other features:

As mentioned earlier, there will be loss of body strength with anorexia. The food ingested will not be digested properly; in view of these there will be defective haemopoiesis. The *Pitha (Ranjitha pitha)* which impart colour to the skin will also become hypovolumic and the *Pitha dosha* will aggravate; as a result the activities of the other doshas will be adversely affected; the potency of directional factor is also decreased and aggravates the disease. As the disease advances, *Kapha* also will increase. In addition, swelling of the body will also occur.

Pulse: If *Sethuma* pulse is altered it may denote *Pandu.*

3.4. MODERN ASPECT OF THE DISEASE

Anaemia:

Anaemia is a decrease in number of red blood cells (RBCs) or less than the normal quantity of haemoglobin in the blood. However, it can include decreased oxygen-binding ability of each haemoglobin molecule due to deformity or lack in numerical development as in some other types of haemoglobin deficiency. Because haemoglobin (found inside RBCs) normally carries oxygen from the lungs to the capillaries, anaemia leads to hypoxia (lack of oxygen) in organs. Since all human cells depend on oxygen for survival, varying degrees of anaemia can have a wide range of clinical consequences.

Anaemia is the most common disorder of the blood. The several kinds of anaemia are produced by a variety of underlying causes. It can be classified in a variety of ways, based on the morphology of RBCs, underlying etiologic mechanisms, and discernible clinical spectra, to mention a few. The three main classes include excessive blood loss (acutely such as a haemorrhage or chronically through low-volume loss), excessive blood cell destruction (haemolysis or deficient red blood cell production (ineffective haematopoiesis).

Prevalence of anaemia:

Anaemia is a major world health problem. The prevalence of anaemia has been studied in many populations but it is difficult to compare data from different sources because of variations in methodology and criteria adopted. Certain patterns emerge, however. An early survey carried out in Great Britain established that haemoglobin levels were low in a significant proportion of the population, particularly susceptible groups being children under the age of five years, pregnant women. A later random population study in the United Kingdom reported a prevalence of anaemia of 14% for women aged 55-64 years and 3% for men aged 35-64 years. These and similar studies have shown that anaemia is commonest in women between ages of 15 and 44 years and that it then becomes relatively less frequent, although the prevalence increases again in the 75 year and over age group. Interestingly, it is only in the latter group that the prevalence in males and females is almost the same. Where the cause of the anaemia has been analysed in these surveys, the majority of cases have been due to iron deficiency. No doubt this prevalence data varies considerably between the developed countries, but it clear that nutritional anaemia is relatively common in most populations at certain periods during development and late in life.

Main causes of anaemia due to defective production of red cells:

Reduced proliferation of precursors

- Iron deficiency anaemia
- Anaemia of chronic disorders
  - Infections, malignancy, collagen disease, etc
- Reduced erythropoietin production
  - Renal disease
- Reduced oxygen requirements
  - Hyperthyroidism
  - Hyperpituitrism
- Reduced O_2 affinity of haemoglobin
- Erythropoietin antibody production
- Primary disease of the bone marrow
- Aplastic anaemia
  - Primary
  - Secondary to drugs, irradiation, chemicals, toxins, etc
  - Leukaemia
  - Lymphoma

Defective maturation of precursors

- Nuclear maturation
  - Vitamin B_{12} deficiency
  - Folate deficiency
  - Erythroleukaemia
- Cytoplasmic maturation
  - Iron deficiency
  - Disorders of globin synthesis
  - Disorders of haem and iron metabolism
  - Disorders of porphyrin metabolism
- Unknown mechanism
  - Congenital dyserythropoietic anaemias
  - Myelodysplastic syndrome
  - Infection
  - Toxins and chemicals.

Classification: 1.Anaemia associated with blood loss or increased blood destruction

Causes:

a) Haemorrhage

b) Chronic – Peptic ulcer, Piles, Hookworm anaemia, Purpura,Uterine bleeding

2. Haemolytic diseases causing anaemias

a) Chemical and haemolytic poisons, eg lead, arsenic etc

b) Specific infection – Malaria,septicaemia

c) Abnormal structure of the red cell eg. Acholuric jaundice,splenic anaemia

3. Anaemia due to Defective blood formation

a) Nutritional anaemia (iron deficiency anaemia)

b) Lack or failure of utilization of specific anti anaemic factor, Ex, pernicious anaemia

c) Aplastic anaemia, Failure of the red bone marrow to produced blood cells. Ex. Exposure to radio activity, chemicals and drugs.

Table.1

WHO's Haemoglobin Thresholds used to define Anaemia (1 g/dL = 0.6206 mmol/L)

Age or Gender group	Hb threshold (g/dl)	Hb threshold (mmol/l)
Children (0.5–5.0 yrs)	11.0	6.8
Teens (12–15 yrs)	12.0	7.4
Women, Non-pregnant (>15yrs)	12.0	7.4
Women, pregnant	11.0	6.8
Men (>15yrs)	13.0	8.1

Symptoms:

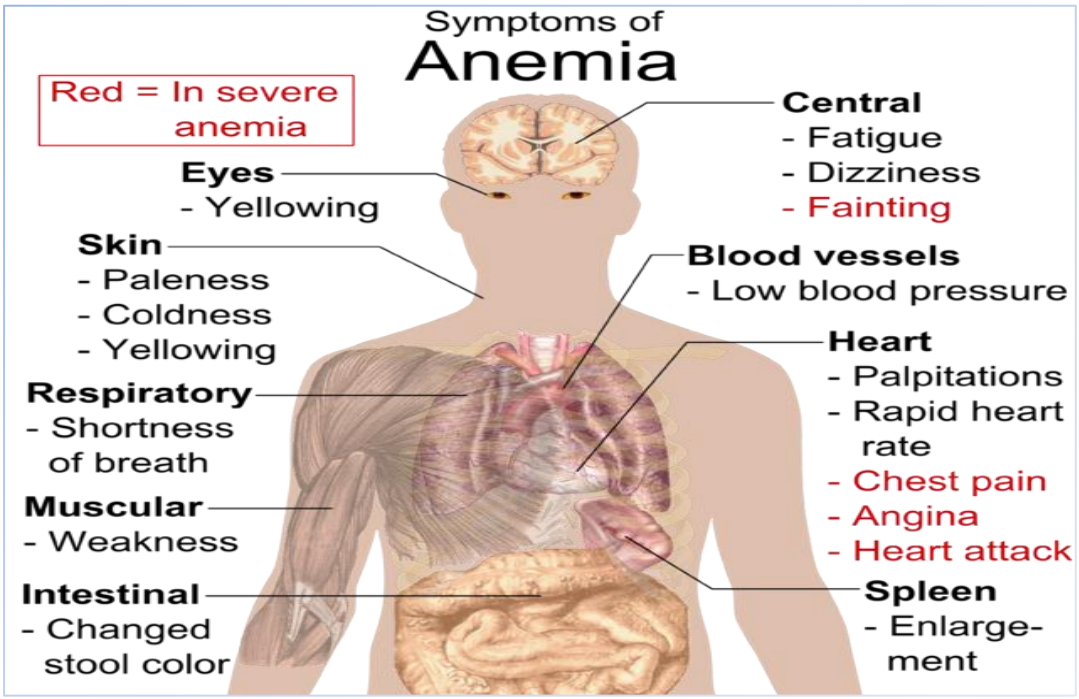

Figure.2 Symptoms of Anaemia

Investigation:

Doctors can easily detect anaemia by drawing a blood sample for a complete blood count. Based on the results of the test and thorough evaluation of the patient, the doctor may order more tests to determine the exact cause of anaemia. The complete blood count may be done as part of a routine general check-up or based upon the presence of signs and symptoms suggestive of anaemia.

Lab tests for anaemia

Complete blood count (CBC):

Determines the severity and type of anaemia (microcytic anaemia or small-sized red blood cells, normocytic anaemia or normal-sized red blood cells, or macrocytic anaemia or large-sized red blood cells) and is typically the first test ordered. Information about other blood cells (white cells and platelets) is also included in the CBC report. Haemoglobin (Hgb) and (Hct) measurements in a complete blood count test are commonly used to diagnose anaemia. They measure the amount of haemoglobin, which an accurate reflection of red blood cell (RBC) quantity in the blood.

Stool haemoglobin test: Tests for blood in the stool may detect bleeding from the stomach or the intestines (stool Guaiac test or stool occult blood test).

Peripheral blood smear: Looks at the red blood cells under a microscope to determine the size, shape, number, and appearance as well as evaluate other cells in the blood.

Iron level: A serum iron level may tell the doctor whether anaemia may be related to iron deficiency or not. This test is usually accompanied by other tests that measure the body's iron storage capacity, such as transferrin level and ferritin level.

Transferrin level: Evaluates a protein that transports iron in the body.

Ferritin: Evaluates at the total iron available in the body.

Folate: A vitamin needed to produce red blood cells, which is low in people with poor eating habits.

Vitamin B12: A vitamin needed to produce red blood cells and low in people with poor eating habits or in pernicious anaemia.

Bilirubin: Useful to determine if the red blood cells are being destroyed within the body which may be a sign of haemolytic anaemia.

Lead level: Lead toxicity was formerly one of the more common causes of anaemia in children.

Haemoglobin electrophoresis: Sometimes used when a person has a family history of anaemia; this test provides information on sickle cell anaemia or thalassemia.

Reticulocyte count: A measure of new red blood cells produced by the bone marrow

Liver function tests: A common test to determine how the liver is working, which may give a clue to other underlying disease causing anaemia.

Kidney function test: A test that is very routine and can help determine whether any kidney dysfunction exists. Kidney failure can result in erythropoietin (Epo) deficiency, leading to anaemia.

3.5. LATERAL RESEARCH

Anti oxidant, Anti microbial and cytotoxic activities of selected medicinal plants from Yemen. (MAPA) February 2008. Volume 30. No.1

Ninety crude extracts, including dichloromethane, methanol and aqueous extracts from 30 medicinal plants used in the Yemeni ethanomedicine to treat common infections, were screened in vitro for antimicrobial activities. Most of the plants showed antibacterial activities, Extracts from *Tamarindus indica* flowers and *Ficus vasta* fruits have been the most active of the 30 plants tested 13 showed antifungal activity (40percent) against one

or more human pathogenic fungi. The strongest inhibition was exhibited by *Azima tetracantha* (fruits) *Sansevieria ehrenbergii* (fruits), and *Solanum incanum*(fruits) ten methanol extracts especially those of acacia asak barks and *solanum nigrum* fruits,showed effective free radical scavenging activities in the DPPH assay.remarkable cytotoxic activity against FL – cells was shown only for 5 plants among them *Plicosepalus curviflorus* (stems).

Screening of Antimutagenicity via Antioxidant activity in Cuban medicinal plants. October 2004, MAPA. Volume 26. No.5

The reducing activity on the 1,1 – diphenyl 1-2 –picrylhydrazyl(DPPH) radical, oh radical scavenging potential, in vitro inhibition of lipid peroxidation and modulation of mutagenicity induced by terbutylhydroperoxide (TBH) in Escherichia coli were sequentially screened in 45 species of plants used with medicinal purposes in cuba, in a search for antioxidant agents which protect DNA against oxidative stree. Five species, e.g *Tamarindus indica, Lippa alba, Pimentia dioica, Rheedia aristata* and *Curcuma longa* displayed IC50 less than 30microg/ml in the DPPH radical reduction assay and IC50 less than 32microg/ml in lipid peroxidation inhibition testing. *Pimenta diocia* and *curcuma longa* showed also a 20 percent inhibition of the in vitro induced OH attack to deoxyglucose, Eugenol, the main constituent of the essential oil of *Pimenta dioica*, also inhibited oxidative mutagenesis by TBH in Escherichia coli, at concentration ranging from 150 to 400 µg/plate.

A review on *Artemisia absinthiumand, Tamarindus indica* in the manangement of jaundice. MAPA 2011 June.**Volume 33.No.3**

Jaundice is a disease, which is characterized by yellowish or blackish discoloration of skin. The cause of which is the accumulation of yellow or black humour in the cutaneous tissues and its nearby tissues.usually this humour is free of putrefaction. Based upon the nature of discoloration jaundice is classified into 2 types. 1.) yarqaan – e- asfar (yellow jaundice). 2) Yarqaan – e- aswad (black jaundice). This type of jaundice does not occur due to excess of bile. Hence it is not discussed here. In yarqaan -e –asfar (yellow jaundice) there is an abnormal excess flow of bile towards the circulatory system,ie, inside the blood cause discoloration of the entire skin, conjunctiva and other secretions and organs of the body. Benefits and authenticity of two drugs. Afsanteen and tamare – Hindi are described which are use for centuries to treat Yarqaan (jaundice) in Unani system of Medicine.

4. MATERIALS AND METHODS

4.1. Preparation of *chooranam:*

The *Tamarindus indicus (Puli Ilai)* leaves 10kg of fresh leaves were taken, then it undergone for shade dry after that the net weight of the leaves were around 3kg.

Collection and authentication of the materials:

The Tamarind leaves used in this study were collected from *Vadalur, Cuddalore (dt), TamilNadu,* India during the month of May 2012. The collected leaves were authenticated by the *Gunapadam* experts in Department of *P.G Gunapadam Govt.Siddha Medical College, Arumbakkam Chennai – 106* and Botanist, the Director Plant Anatomy Research Centre, Chennai. One specimen sample has been preserved in the Department for future reference.

Purification of the Raw Drug:

The collected leaves were washed in running tap water and dried in shade.

Preparation of the *Chooranam:*

The preparation was taken from ***Gunapadam – Mooligai*** written by *K.S.Murugesa Mudhaliyar*. The dried Tamarind leaves were grounded into a fine powder. The powder was sieved through a clean white cloth to get a uniform particle size of *Chooranam.*

Purification of *Chooranam:*

The prepared *chooranam* was purified by a process called *Pittaviyal*. For this process cow's milk and water were taken in equal ratio and half filled in filled mud pot. A clean dry cloth was tied firmly around the mouth of the mud pot. *Chooranam* was placed over the tied cloth. Another mud pot of similar size was kept over the mouth of the mud pot. The gap between mud pots was tied with a wet cloth to avoid evaporation. The mud pot was kept on fire and boiled until the cow's milk reduced to the lower pot. Then the *chooranam* was taken, dried, powdered finely.

Preservation:

The purified *Chooranam* was stored in a clean, air tight glass container. Since the life period of the *Chooranam* is only three month, the prepared *Chooranam* must be used within this period.

Figure.3. *Tamarindus indica (Puli ilai)*

Figure.4. *Puli ilai chooranam (Tamarindus indica)*

Administration of the drug:

Form of the medicine	: *Chooranam*
Route of Administration	: Enteral
Dose	: 1gm
Anubanam (Vehicle)	: Honey
Times of Administration	: Two times a day; before food
Duration	: 48 Days.

4.2. STANDARDIZATION OF THE DRUG

4.2.1. Pharmacognostic aspect:

Macroscopically:

Organoleptic evaluation:

Organoleptic evaluation refers to evaluation of the formulation by Colour, Odour, Taste and texture etc. The Organoleptic characters of the sample were evaluated as per the reference of (Siddiqui et *al* 2002*).*

Microscopically:

Collection of specimens:

The plant specimens for the proposed study were collected from Chennai. Care was taken to select healthy plants and normal organs. The required samples of different organs were cut and removed from the plant and fixed in FAA (Farmalin-5ml+ Acetic acid-5ml + 70% Ethyl alcohol-90ml).After 24 hrs of fixing, the specimens were dehydrated with graded series of tertiary –Butyl alcohol as per the schedule given by Sass, 1940. Infiltration of the specimens was carried by gradual addition of paraffin wax melting point 58-60 C) until TBA solution attained super saturation. The specimens were cast into paraffin blocks.

Sectioning:

The paraffin embedded specimens were sectioned with the help of Rotary Microtome. The thickness of the sections was 10-12 μm. Dewaxing of the sections was by customary procedure (Johansen, 1940). The sections were stained with Toluidine blue as per the method published by O'Brien et al. (1964). Since Toluidine blue is a polychromatic stain. The staining results were remarkably good; and some cytochemical reactions were also obtained. The dye rendered pink colour to the cellulose walls, blue to

the lignified cells, dark green to suberin, violet to the mucilage, blue to the protein bodies etc. wherever necessary sections were also stained with safranin and Fast-green and IKI(for Starch).

For studying the stomatal morphology, venation pattern and trichome distribution, paradermal sections (sections taken parallel to the surface of leaf) as well as clearing of leaf with 5% sodium hydroxide or epidermal peeling by partial maceration employing Jeffrey's maceration fluid (Sass, 1940) were prepared. Glycerine mounted temporary preparations were made for macerated/cleared materials. Powdered materials of different parts were cleared with NaoH and mounted in Glycerine medium after staining. Different cell component were studied and measured.

Photomicrographs:

Microscopic descriptions of tissues are supplemented with micrographs wherever necessary. Photographs of different magnifications were taken with Nikon lab photo 2 microscopic Unit. For normal observations bright field was used. For the study of crystals, starch grains and lignified cells, polarized light was employed. Since these structures have refrigerant property, under polarized light they appear bright against dark background. Magnifications of the figures are indicated by the scale-bars. Descriptive terms of the anatomical features are as given in the standard Anatomy books (Esau, 1964). The result showed in Figure :5 and Figure: 6

4.2.2. PHYSICO-CHEMICAL ANALYSIS

ASH AND ACID INSOLUBLE ASH:

To the ash add 1:5 Hcl: Distilled water 15 ml boil, cooled and then filtered using Whatman filter paper (No.41) repeat 3 to 4 times till the yellow colour disappear or colourless, then remove the filter paper and add to the filter to the original dish and keep it in the muffle furnace at 600^{o} C and take constant weight and calculate the acid insoluble ash value.

$$\text{Acid insoluble ash (\%)} = \frac{\text{Weight of acid insoluble residue x 100}}{\text{Weight of the sample}}$$

Acid insoluble residue = Acid insoluble ash value – Empty weight of the dish

Loss on drying:

3gm of the drug is heated in a hot oven at 105^0 c to constant weight. The % of weight was calculated.

Loss on drying value at 105^0 c - 10.96 %w/w

Potential of hydrogen (ph):

The pH scale is logarithmic and runs from 0.0 to 14.0 with 7.0 being neutral. Readings less than 7.0 indicate acidic solutions, while higher readings indicate alkaline or base solutions.

Above mentioned Quantitative analysis results are showed in the table: 12

Thin layer chromatography:

Extract Preparation:

4 gram of the *chooranam* was soaked overnight in chloroform. Boiled on a water bath for 10 minutes, filtered and concentrated to 10 ml.

Procedure:

The extract was applied on the TLC using capillary and developed in the solvent system. The developed TLC plate was air dried and photograph was taken in white light, then dipped in vanillin-sulphuric acid reagent, heated in an oven at 105°C until the development of coloured spots and photograph taken (Figure. 7) TLC results are showed in Table 14.

4.2.3. QUALITATIVE PHYTOCHEMICAL ANALYSIS:

Materials and methods:

The leaves and aerial part of *Tamarindus indica (L)* were destalked, washed and shade dried exposing the leaves for desiccation. The leaves were later milled to obtain the fine powder using an electric blender. The yield of extract was calculated. Photochemical screening procedures carried out were adopted from this analysis determines the biologically active compounds that contribute to the flavour, colour and other characteristics of vegetable leaves.

S.I.No	EXPERIMENT	OBSERVATION	INFERENCE
1.	**Test for alkaloids:** (Dragendorff's Test) Few mg of extract in separate test tube was warmed with 2% Sulphuric acid for 2 minutes. And it was filtered in separate test tube and few drops of Dragendorff's reagent were added.	The absence of orange red precipitates indicates the presence of alkaloids.	Absence of alkaloids.
2.	**Triterpenoids (Noller's Test)** To fewmg of extract, add tin and thionyl chloride and heat in water bath. Purple colour indicates the presence of tritepenoids.	Purple colour indicates.	Presence of triterpenoids.
3.	**Test for Flavonoids (Shinoda test):** Substance is dissolved in alcohol, added with magnesium bits and concentrated hydrochloric acid.On heating over a water bath,	The appearance of majenta colour..	Presence of flavonoids.
4.	**Test for Saponins** To few mg of extract distilled water is added and shaken well.	The formation of foam indicates	Presence of saponin.
5.	**Test for Steroids (Lieberman Burchard Test)** To few mg of the extract 2 ml of chloroform is added in a dry test tube. Few drops of acetic acid is added, heated and few drops of acetic anhydride and 2 drops of concentrated sulphuric acid are added.	The green colour indicates	The presence of steroid.
6.	**Test for Proteins (Biuret test)** To the sample solution in a test tube, add sodium hydroxide solution and then add a few drops of very dilute (1 %) copper II sulphate solution and mix gently.	Appearance of purple colour indicates.	The presence of protein.
7.	**Test for Cardiac glycoside (Keller-Killani Test).** Add 2 ml of glacialacetic acid containing a drop of ferric chloride solution and and 0.5 ml of concentrated sulphuric acid to the chloroform extract of the plant.	The blue colour in the acetic acetic acid layer shows	The presence of cardiac glycosides.
8.	**Test for Acids:** Extract is treated with sodium bicarbonate solution.	Effervescence shows.	The presence of acid.

Table.2. Phyto chemical analysis.

Phytochemical screening:

Chemical tests were carried out using the aqueous extracts from Plants and or the powdered specimens, using standard procedures to identify the constituents as described by Sofowara (1993), Trease and Evans(1989) and Harborne(1973). The results are showed in Table: 13

4.2.4. Bio -chemical analysis:

Preparation of extract of test drug:

Add 5 gm of ***Puli ilai chooranam*** to 50ml of distilled water. The solution is boiled for 20 minutes, and then it is cooled and then filtered in a 100ml volumetric flask. Use the Extract for the following tests.

S.NO	EXPERIMENT	OBSERVATION	INFERENCE
1.	**Test for Reducing Sugar :** To 5ml of Benedicts qualitative reagent, add 10 drops of extract, then boil for two minutes	Presence of Green Precipitate	Presence of Reducing Sugar
2.	**Test for Starch :** To 5 ml of extract add 2ml of acetic acid and then add few drops of N/50 Iodine Solution.	Presence of of Blue Colour	Presence of of Starch
3.	**Test for Proteins :** To 2 ml of extract, add 2ml of 5% Sodium Hydroxide mix and add 2 drops of Copper Sulphate Solution.	Presence of Violet Colour	Presence of of Proteins
4.	**Test for amino Acid :** Place 2 drops of extract on a filter paper and allow to dry well. Then spray 1% ninhydrin over the same and allow to dry.	Presence of Violet Colour	Presence of of Amino Acid
5.	**Test for Albumin :** To 2 ml of extract, add 2ml of Esboch's reagent.	Presence of Yellow Precipitate	Presence of Albumin
6.	**Test for Phosphate :** To 2ml of extract, add 2ml of ammonium Molybdate solution and 2ml of concentrated Nitric Acid.	Presence of Yellow Precipitate	Presence of Phosphate
7.	**Test for Sulphate :** To 2 ml of extract add 2ml of 4% ammonium oxalate solution.	Presence of White Precipitate	Presence of Sulphate

S.No	EXPERIMENT	OBSERVATION	INFERENCE
8.	**Test for Chloride :** Add 2ml of extract to dilute nitric acid till the effervescence ceases. Then add 2 ml of Silver Nitrate Solution.	Presence of Cloudy White Precipitate	Presence of Chloride
9.	**Test for Iron :** To 2ml of extract, add 2ml of ammonium thiocynate solution and add 2ml of concentrated Nitric Acid.	Presence of Red Colour	Presence of Iron
10.	**Test for Calcium :** To 2 ml of extract, add 2 ml of 4% ammonium Oxalate Solution.	Presence of White Precipitate	Presence of Calcium
11.	**Test for Sodium :** Make a paste with 2 pinches of the sample with Hcl and Introduce it into the blue flame.	Absence Yellow Flame	Absence of Sodium
12.	**Test for Potassium :** Add a pinch of the sample to 2 ml of Sodium Nitrate Solution. Then add 2ml of Cobal Nitrate in 20% acetic acid.	Absence Yellow Precipitate	Absence of Potassium
13.	**Test for Zinc :** To 2ml of extract, add few drops of Sodium Hydroxide.	Absence White PPT	Absence of Zinc
14.	**Test for Magnesium :**To 2ml ofextract, add few drops of Sodium Hydroxide Solution	Presence of White PPT	Presence of Magnesium
15.	**Test for Alkaloids :** a. To 2ml of extract, add 2ml of Potassium Iodide Solution b. To 2ml of extract add 2ml of Picric Acid. c. To 2 ml of extract add 2ml of Phosphotunqstic Acid.	 Absence Red Colour. Absence Yellow Colour. Absence White Precipitate.	 Absence of Alkaloids Absence of Alkaloids Absence of Alkaloids
16.	**Test for Tannic Acid :**To 2ml of extract add 2 ml of Ferric Chloride Solution	Presence of Black Precipitate.	Presence of Tannic Acid

Table.3 Bio – Chemical analysis

4.3. TOXICOLOGICAL STUDY

Materials and methods:

Acute toxicity:

Acute oral toxicity test for the *Puli ilai Chooranam* was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice. The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal.

Observation of toxicity signs:

General behaviour, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded. The results are showed in Table.No.15

4.4. PHARMACOLOGICAL STUDY:

Haematinic Activity of *Puli ilai chooranam* in phenylhydrazine induced anaemic rats

Materials and methods:

Drug and Stock solution

The *Puli Ilai Chooranam* was prepared as per the procedure in traditional Siddha text recommendation and made into suspension using CMC as a suspending agent and used in this study. The resulting suspension was then grounded and filtered. The filtrate was stored in a refrigerator until use. The suspension was further diluted with 2% CMC so as to achieve 200mg/ml stock concentration.

Animals

Male albino rats (150-180g) and Mice of either sex weighing 25-30g were used for the study. The rats were housed in wire-mesh cages with a 12 h light/dark cycle. They had continuous access to food and water during the entire period of experimentation. (Approval number: XIII/VELS/PCOL/44/2000/CPCSEA/IAEC/08.08.2012).

Evaluation of Haematinic Activity

Six rats were kept as normal control group (Group 1), while 24 rats were made anaemic by oral intubations of phenylhydrazine (10 mg/kg body weight) daily for seven days. Rats that developed anaemia with haemoglobin concentration <14 g/dl were recruited for the study. Anaemic rats were randomly divided into 5 groups (2 to 6) and treated as follows: Group 1: received distilled water (1 ml) daily (normal control), Group 2: received 2% CMC (1 ml) daily (anaemic control), Group 3: received oral single dose of the *Puli Ilai Chooranam* 100 mg/kg body weight/day Group 4: received oral single dose of the *Puli Ilai Chooranam* 200 mg/kg, Group 5: received oral single dose of the *Puli Ilai Chooranam* 400 mg/kg Group 6: received oral single dose of the heamatinic syrup 2ml/kg body weight/day. The treatment was continued for 2 weeks.

Haematological investigation

Before and after treatment with drug *Puli Ilai Chooranam* blood was collected from the retro orbital vein of experimental animals after an overnight fast (T=0) and after 1 and 2 weeks of treatment with *Puli Ilai Chooranam*, was used for the determination of red blood cell count (RBC), haemoglobin (Hb) concentration and packed cell volume (PCV). The mean cell volume (MCV), mean cell haemoglobin (MCH) and the mean cell haemoglobin concentration (MCHC) were calculated.

Statistical analysis

Experimental data was analysed using analysis of variance (ANOVA) and Dunnet's't' test to determine significant differences between means. The statistical analysis system (INSTAT-V3) package was used for this analysis.

4.5. Clinical assessment:

At the present time Life style changed the food habits. This condition made significant impact in changing the human physiology into pathology. Which leads to Haematalogical disorders are emerged although there is a lot of medications available for this disease, still there is a thrive for less adverse effect drugs. Herbal medicines are playing vital role on curing diseases without marked adverse effects even though on long term intake. From this plant kingdom I have selected this herb which proved its Haematinic activity pre clinically. *Puli ilai chooranam*, an herbal medicine was used for this clinical trial to prove its safety and efficacy against *Pandu noi*.

Objectives:

The study was conducted on Anaemia patients to assess the "Haematinic" activity of ***"Puli ilai chooranam"*** clinically, both in-patients and out-patients of both sex and varying age groups.

Study centre:

The clinical study for Anaemia was carried out in outpatient department and in patient department of Govt. Siddha Medical College Hospital Arumbakkam Chennai.

Design of the study:

Open clinical trial, Phase II B

Selection:

50 patients from both sexes of various age groups were selected for clinical trial. Among 50 patients 40 patients were treated as out-patients, 10 patients were treated as in patients. The selection was based on the including and excluding criteria. They were clinically diagnosed on the basis of *Siddha* principles with modern laboratory findings.

Registration process

To register a patient, the following documents were completed by the investigator.

- Copy of required laboratory tests
- Signed patient consent form
- Other appropriate forms (e.g., Trial proforma).

This Clinical trial is an ethical and scientific quality standard for designing, conducting and recording trials that involve the participation of human subjects. Compliance with this standard provides assurance to public that the rights, safety and well being of trial subjects are protected, consistent with the principles enshrined in the Declaration of Helsinki and ensures that clinical trial data are credible.

Inclusion criteria:

- Hb – 7 to 10 gm%
- Pallor of conjunctiva and nail beds.
- Loss of appetite
- Ulceration of mouth
- Lassitude
- Emaciation
- Palpitation
- Dyspnoea on exertion
- Fatigue
- Breathlessness
- Tiredness
- Age 20 to 60 yrs
- Patient willing to attend the op once in 7 days or willing to be admitted in the IP for 30 to 45 days

Exclusion criteria:

- Chronic liver failure
- Chronic renal failure
- Myxoedema
- Thalassemia
- Worm Infestation

Withdrawal Criteria:

- ❖ Any other acute severe illness
- ❖ Drug intolerance

Termination Criteria:

- ❖ Voluntary termination
- ❖ Not reporting subsequently.

Clinical Pathological Examination:

Blood Test:

- ❖ Total count
- ❖ Differential count
- ❖ Haemoglobin
- ❖ Erythrocyte sedimentation rate
- ❖ Blood sugar
- ❖ Blood urea
- ❖ MCV - Mean Corpuscular Volume
- ❖ PCV - Packed Cell Volume

URINE EXAMINATION:

- ❖ Albumin
- ❖ Sugar
- ❖ Deposits.

MOTION EXAMINATION:

- ❖ Ova
- ❖ Cyst
- ❖ Occult blood

Administration of the drug:

Form of the medicine	: *Chooranam*
Route of Administration	: Enteral
Dose	: 1gm
Anubanam (Vehicle)	: Honey
Times of Administration	: Two times a day; before food
Duration	: 48 Days.

Diet and Medical advice:

A diet rich in iron helps in increasing the haemoglobin levels. It is seen that iron from non-vegetarian sources (haeme-iron) is more readily absorbed than iron from vegetarian sources (non-haeme iron). The addition of vitamin C to the diet helps in the absorption of iron and hence squeezing some lime juice on your salads/food and eating or drinking foods rich in vitamin C along with your meals would facilitate iron absorption. Avoid drinking tea with your meals as the tannins present in the tea can interfere with the absorption of iron from the meal.

Adults and children who are diagnosed of low haemoglobin count require an iron rich diet. Diet for anaemia contains iron rich veg/non-veg sources and is nutritionally rich in vitamins and folic acid.

Do's:

1. Eat more iron rich foods. The very best sources of iron that is easily absorbed by the human body are:

- Meat (especially organ meats like liver and kidneys)
- Fish
- Eggs (especially the yolk)
- Cheese

Commercial breakfast cereals are fortified with easily absorbable iron so they can also make a good contribution.

2. Vitamin C improves iron absorption. Have a glass of fresh orange juice with your breakfast which could contain egg, the breakfast cereals, or a bit of fried liver.

3. You also need folic acid to assist in preventing Anaemia. Folic acid is mainly found in green leafy vegetables, and you can also obtain a supplement if necessary.

Dont's:

1. Don't go on a vegetarian diet without consulting your doctor and dietician.

2. Avoid iron-containing cocktails of vitamins since these generally do not contain enough iron and are expensive.

3. Drugs that reduce acid production by the stomach such as Cimetidine (Tagamet) may inhibit iron absorption and these tablets should not be taken simultaneously.

4. There are no quick fixes. It is necessary to continue iron therapy for four to six months to correct the Anaemia and replenish stores.

Trial conduct:

This study was conducted in compliance with the protocol approved by the Institutional Review Board, and according to Good Clinical Practice standards. No deviation from the protocol was implemented without the prior review and approval of the IRB except where it may be necessary to eliminate an immediate hazard to a research subject. In such case, the deviation was reported to the IRB as soon as possible.

Follow up:

Assessment was taken for every three days before treatment and after treatment. During this period clinical assessment and laboratory investigation was carried out.

Statistical analysis:

The data were tabulated and analyzed by students 'T' test. The results were showed in Table 18 and 19.

Ethical Review:

The protocol and amendments were submitted to the Govt siddha medical college, Institutional Ethical Committee (IEC) and got formal approval for conducting the study. The decision of the IEC concerning the conduct of the study was made in writing to the investigator.All subjects for this study was provided a consent form describing this study and provided sufficient information for subjects to make an informed decision about their participation in this study. This consent form was submitted with the protocol for review and approval by the IEC. The formal consent of a subject, using the IEC-approved consent form, was obtained before that subject is submitted to any study procedure. This consent form was signed by the subject or legally acceptable surrogate, and the investigator-designated research professional obtaining the consent.

The results of the Clinical trial are showed in Table: 16.

CLINICAL STUDY ON PULI ILAI CHOORANAM IN - OUT PATIENTS DEPT. IN THE MANAGEMENT OF ANAEMIA

Sl. No.	O.P. No.	Name	Age/ Sex	HAEMATOLOGICAL REPORT														URINE ANALYSIS						STOOL EXAMINATION			
				BEFORE TREATMENT				AFTER TREATMENT				ESR (mm)				Hb(Gm)		BT			AT			BT		AT	
				TC CU/mm	DC			TC CU/mm	DC			BT		AT		BT	AT	Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst
					P	L	E		P	L	E	1/2 hr	1 hr	½ hr	1hr													
1.	3114	MOORTHY	70/M	9000	53	41	6	9100	58	38	4	23	52	12	25	11.0	13.2	NIL	++	Opc	NIL	++	NIL	NIL	NIL	NIL	NIL
2.	6754	DEVI	32/F	9800	59	36	5	9900	62	34	5	45	88	40	62	10.2	12.7	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL	NIL	NIL
3.	6865	DEENA	17/F	6100	44	51	5	6300	51	44	5	35	72	20	40	5.6	9.2	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL	NIL	NIL
4.	3345	RAMESH	38/M	9800	59	36	5	9800	59	37	4	10	25	8	22	13.1	16.2	NIL	NIL	Fpc	NIL	NIL	Fpc	NIL	NIL	NIL	NIL
5.	5087	RAMASWAMY	56/M	9200	52	39	9	9200	58	38	4	12	30	10	15	9.0	11.5	NIL	NIL	Oec	NIL	NIL	NIL	NIL	NIL	NIL	NIL
6.	3241	RAJATHI	55/F	7600	49	46	5	8000	52	44	4	10	20	10	22	9.0	11.6	NIL	+	Opc	NIL	+	Opc	NIL	NIL	NIL	NIL
7.	2396	NARAYANAN	60/M	9400	57	36	7	10100	60	36	4	10	16	5	10	11.0	11.5	NIL	+	Opc	NIL	+	NIL	NIL	NIL	NIL	NIL
8.	4566	MURALIRAJ	50/M	8700	59	35	6	8800	58	56	6	2	7	5	15	13.0	14.2	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
9.	6444	PANGAJAM	47/F	10400	63	31	6	10400	62	33	5	40	75	20	40	13.0	14.5	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
10.	6373	SARANYADEVI	16/F	7600	48	46	6	7800	50	45	5	8	10	10	12	7.0	10.8	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
11.	3674	BHAVANI	12/F	9000	49	45	6	9200	50	45	5	10	18	10	15	12.0	14.2	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
12.	8062	PONNUTHAYE	70/F	7400	53	41	6	7600	52	40	8	50	71	30	60	8.0	10.4	NIL	++	NIL	NIL	++	NIL	NIL	NIL	NIL	NIL
13.	9665	SATHYAN	44/M	9400	57	38	5	9500	59	36	5	4	9	10	15	10.2	12.7	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
14.	5702	SREE KIRIJA	41/F	10100	60	34	6	10200	60	36	4	42	75	20	40	8.0	11.1	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL	NIL	NIL
15.	9900	CHILAMBARASI	60/F	7800	55	38	7	7900	54	42	4	30	62	15	30	9.0	11.2	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
16.	2055	JAYAPAL	44/M	9800	55	39	6	9800	55	41	4	9	15	8	10	14.3	16.2	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
17.	8236	HARIHARAN	22/M	9700	60	36	4	9800	60	35	5	3	10	4	12	13.4	15.1	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
18.	8270	PERIASAMY	40/M	8600	55	39	6	8700	56	38	6	14	27	10	20	13.0	16.4	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
19.	4200	ARIVAZHAGI	54/F	8000	55	39	6	8000	57	37	6	35	62	20	40	10.0	12.4	NIL	+	NIL	NIL	+	NIL	NIL	NIL	NIL	NIL
20.	3838	SAVITHA	19/F	9200	61	36	3	9300	62	35	3	33	60	17	30	12.0	14.3	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL

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				TC CU/mm	DC			TC CU/mm	DC			BT		AT		BT	AT	Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst
					P	L	E		P	L	E	1/2 hr	1 hr	½ hr	1hr													
21.	3322	SREESELVI	37/F	9400	55	32	13	9500	57	33	10	11	24	8	14	10.2	12.1	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
22.	3272	JAMUNA	11/F	7600	48	46	6	7800	52	43	5	8	10	10	12	10.6	12.0	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
23.	1243	RATHA	35/F	10400	66	29	5	10400	65	30	5	25	55	18	40	10.8	13.1	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL	NIL	NIL
24.	7399	SHANTHI	40/F	9200	53	40	7	9200	53	42	5	20	45	15	30	11.0	13.1	NIL	NIL	Fpc	NIL	NIL	Fpc	NIL	NIL	NIL	NIL
25.	3478	GOPALAN	55/M	9100	57	30	13	9400	55	33	12	5	9	5	11	9.0	11.9	NIL	++	Opc	NIL	++	NIL	NIL	NIL	NIL	NIL
26.	9867	DHAMODHARAN	40/M	9800	59	37	4	9400	60	33	7	2	5	2	4	13.0	14.7	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
27.	9666	LALITHA	45/F	10700	63	34	3	10700	62	33	5	38	64	18	34	10.7	12.0	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
28.	3632	LOGESHWARAN	11/M	8000	49	47	4	8100	52	45	3	13	30	8	10	10.6	12.7	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
29.	3988	ABILASH	12/M	9000	55	30	15	9100	58	38	4	10	18	8	10	11.0	13.1	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
30.	4467	UMA	40/F	8400	58	36	6	8400	58	38	4	25	74	15	32	9.0	11.3	NIL	NIL	1-2PC	NIL	NIL	NIL	NIL	NIL	NIL	NIL
31.	2234	INDHIRA	70/F	10000	62	34	4	10100	58	35	7	15	25	8	15	12.0	14.3	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
32.	7546	GOMATHY	32/F	9700	57	38	5	9600	58	37	5	15	45	15	30	11.0	12.6	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
33.	6234	NAGAMMAL	40/F	9600	59	36	5	9600	58	40	2	27	55	20	40	13.0	14.6	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
34.	6789	PRAVEENRAJ	14/M	9000	47	48	5	9100	50	45	5	10	20	10	18	11.0	13.0	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
35.	3456	MAHALAKSHMI	24/F	9400	55	39	6	9500	58	36	6	20	36	18	30	10.4	12.6	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL	NIL	NIL
36.	1023	KAVIYA	35/F	9000	55	42	3	9200	57	40	3	10	18	10	17	12.0	14.4	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
37.	2874	AISHWARYA	32/M	9000	55	39	6	9100	54	40	6	18	35	15	30	12.0	14.7	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
38.	8236	SENTHIL KUMAR	29/M	9400	55	41	4	9500	55	42	3	10	22	12	20	13.0	14.7	NIL	NIL	Fec	NIL	NIL	NIL	NIL	NIL	NIL	NIL
39.	8345	RAMYA	29/F	8000	58	37	5	8100	59	37	4	11	26	8	15	10.0	12.4	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
40.	5567	THURAI	48/M	8800	59	38	3	8900	59	39	2	5	10	6	15	12.0	16.0	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL

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				TC CU/mm	DC			TC CU/mm	DC			BT		AT		BT	AT	Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst
					P	L	E		P	L	E	1/2 hr	1 hr	½ hr	1hr													
1.	4239	NAGULAN	70/M	9000	53	41	6	9100	58	38	4	23	52	12	25	11.0	13.2	NIL	++	Opc	NIL	++	NIL	NIL	NIL	NIL	NIL
2.	6543	SOUNDARYA	32/F	9800	59	36	5	9900	62	34	5	45	88	40	62	10.2	12.7	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL	NIL	NIL
3.	9876	DARSINI	17/F	6100	44	51	5	6300	51	44	5	35	72	20	40	6.0	9.2	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL	NIL	NIL
4.	3543	ZAHEER	38/M	9800	59	36	5	9800	59	37	4	10	25	8	22	13.1	16.2	NIL	NIL	Fpc	NIL	NIL	Fpc	NIL	NIL	NIL	NIL
5.	5087	RAMANAN	56/M	9200	52	39	9	9200	58	38	4	12	30	10	15	9.0	11.5	NIL	NIL	Oec	NIL	NIL	NIL	NIL	NIL	NIL	NIL
6.	6534	RAMANI	55/F	7600	49	46	5	8000	52	44	4	10	20	10	22	9.0	11.6	NIL	+	Opc	NIL	+	Opc	NIL	NIL	NIL	NIL
7.	4356	KIRUBAKARAN	60/M	9400	57	36	7	10100	60	36	4	10	16	5	10	11.0	11.5	NIL	+	Opc	NIL	+	NIL	NIL	NIL	NIL	NIL
8.	4654	MURUGAN	50/M	8700	59	35	6	8800	58	56	6	2	7	5	15	13.0	14.2	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
9.	6543	PALANIYAMMAL	47/F	10400	63	31	6	10400	62	33	5	40	75	20	40	13.0	14.5	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL
10.	6654	VANAJA	16/F	7600	48	46	6	7800	50	45	5	8	10	10	12	7.0	10.8	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL	NIL

CLINICAL STUDY ON *PULI ILAI CHOORANAM* IN - OUT PATIENTS

S.No	O.P .No	Name	Duration
1	3114	MOORTHY	1-7-12 to 25-7-12
2	6754	DEVI	5-7-12 to 29-7-12
3	6865	DEENA	6-7-12 to 30-7-12
4	3345	RAMESH	6-7-12 to 30-7-12
5	5087	RAMASWAMY	1.8.12 To 31.8.12
6	3241	RAJATHI	6-7-12 to 30-7-12
7	2396	NARAYANAN	2.8.12 To 30.8.12
8	4566	MURALIRAJ	8-7-12 to 1-8-12
9	6444	PANGAJAM	8.8.12 To 5.9.12
10	6373	SARANYADEVI	10-8-12 to 9-9-12
11	3674	BHAVANI	18-8..12 To 4.9.12
12	8062	PONNUTHAYE	20-8-12 to 8-9-12
13	9665	SATHYAN	22-8-12 to 20-9-12
14	5702	SREE KIRIJA	24-8-12 to 22-9-12
15	9900	CHILAMBARASI	24-8-12 to 24-9-12
16	2055	JAYAPAL	25-8-12 to 25-9-12
17	8236	HARIHARAN	27-8-12 to 26-9-12
18	8270	PERIASAMY	28-8-12 to 27-9-12
19	4200	ARIVAZHAGI	28-8-12 to 14-9-12
20	3838	SAVITHA	28-8-12 to 15-9-12
21	3322	SREESELVI	28-8-12 to 18-9-12
22	3272	JAMUNA	30-8-12 to 25-9-12
23	1243	RATHA	2-9-12 to 1-10-12
24	7399	SHANTHI	2-9-12 to 28-9-12
25	3478	GOPALAN	3-9-12 to 27-9-12
26	9867	DHAMODHARAN	4-9-12 to 25-9-12
27	9666	LALITHA	5-9-12 to 28-9-12
28	3632	LOGESHWARAN	8-9-12 to 30-9-12
29	3988	ABILASH	10-9-12 to 10-10-12
30	4467	UMA	12-9-12 to 12-10-12
31	2234	INDHIRA	15-9-12 to 10-10-12
32	7546	GOMATHY	20-9-12 to 18-10-12
33	6234	NAGAMMAL	25-9-12 to 22-10-12
34	6789	PRAVEENRAJ	28-9-12 to 20-10-12
35	3456	MAHALAKSHMI	1-10-12 to 25-10-12
36	1023	KAVIYA	5-10-12 to 30-10-12
37	2874	AISHWARYA	10-10-12 to 12-10-12
38	8236	SENTHIL KUMAR	12-10-12 to 12-11-12
39	8345	RAMYA	14-10-12 to 14-11-12
40	5567	THURAI	15-10-12 to 20-11-12

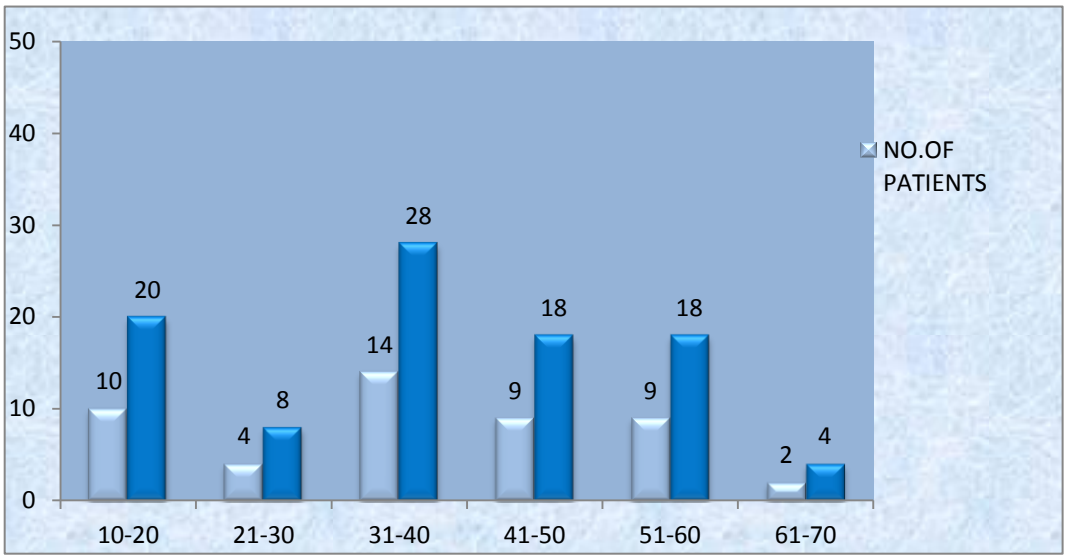
CLINICAL STUDY ON *PULI ILAI CHOORANAM* – IN PATIENTS DEPARTMENT

S.No	OP.No	Name	Duration
1	4239	NAGULAN	5-6-12 to 2-7-12
2	6543	SOUNDARYA	6-7-12 to 20-7-12
3	9876	DARSINI	15-7-12 to 5-8-12
4	3543	ZAHEER	20-7-12 to 5-8- 12
5	5087	RAMANAN	1-8-12 to 25-8-12
6	6534	RAMANI	15-8-12 to 30-8-12
7	4356	KIRUBAKARAN	2-9-12 to 25-9-12
8	4654	MURUGAN	25-9-12 to 15-10-12
9	6543	PALANIYAMMAL	5-10-12 to 25-10-12
10	6654	VANAJA	17-10-12 to 2-11-12

CLINICAL ASSESSMENT

Age wise distribution Table.7.

SL. NO	AGE IN YEARS	NO. OF PATIENTS	PERCENTAGE (%)
1	10-20	10	20
2	21-30	4	8
3	31-40	14	28
4	41-50	9	18
5	51-60	9	18
6	61-70	2	4
TOTAL		50	100



Inference:

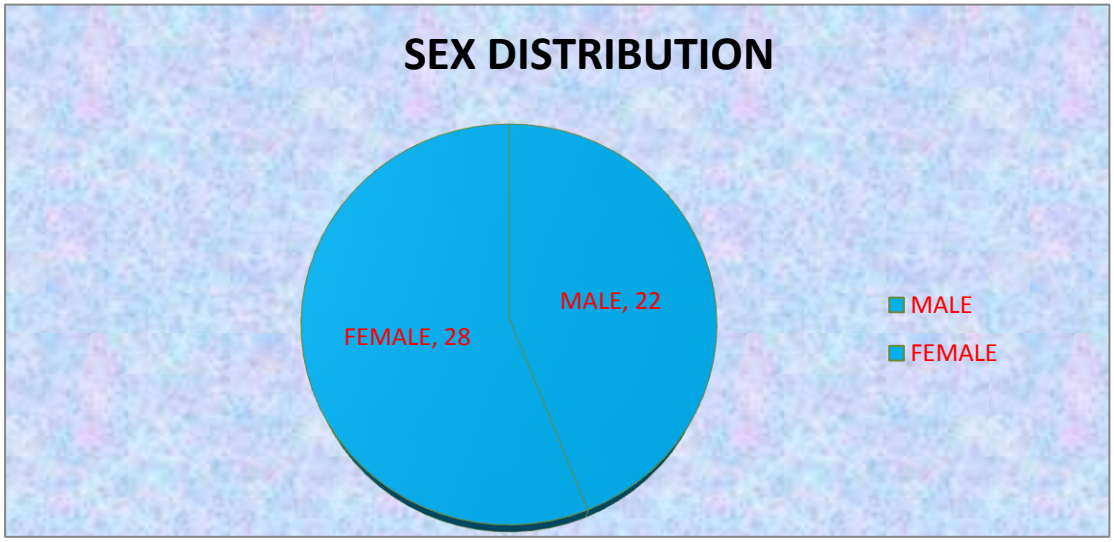
Among 50 patients,

- 10 Patient belongs to the age group of 10-20 years
- 4 Patient belongs to the age group of 21-30 years
- 14 Patients belongs to the age group of 31-40 years
- 9 Patients belongs to the age group of 41-50 years
- 9 Patients belongs to the age group of 51-60 years
- 2 Patients belongs to the age group of 61-70 years

Table.8

SEX DISTRIBUTION

SL. NO	SEX	NO. OF PATIENTS	PERCENTAGE
1	Male	22	44
2	Female	28	56
TOTAL		50	100



INFERENCE:

Among 50 patients,

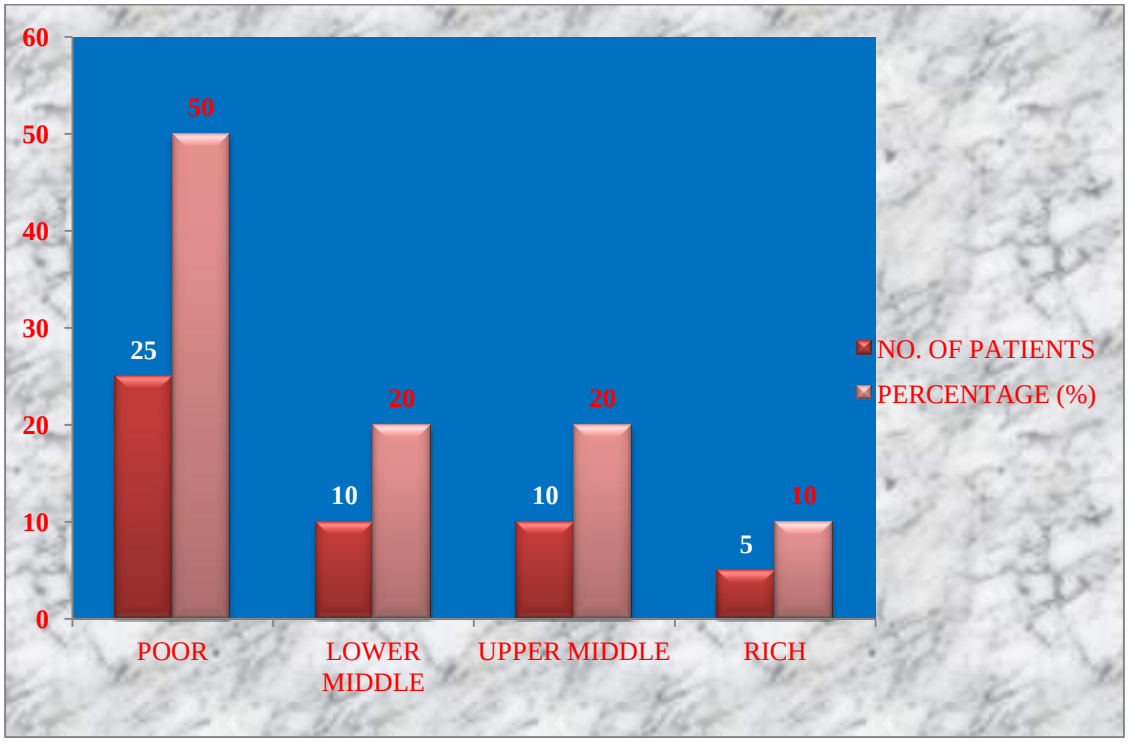
- 22patients were Male
- 28 patients were Female

Table.9.

SOCIO-ECONOMIC STATUS

SL. NO	SOCIO – ECONOMIC STATUS	NO. OF PATIENTS	PERCENTAGE (%)
1	Poor	25	50
2	Lower middle	10	20
3	Upper middle	10	20
4	Rich	5	10
TOTAL		50	100

SOCIO-ECONOMIC STATUS



INFERENCE: Among 50 patients,

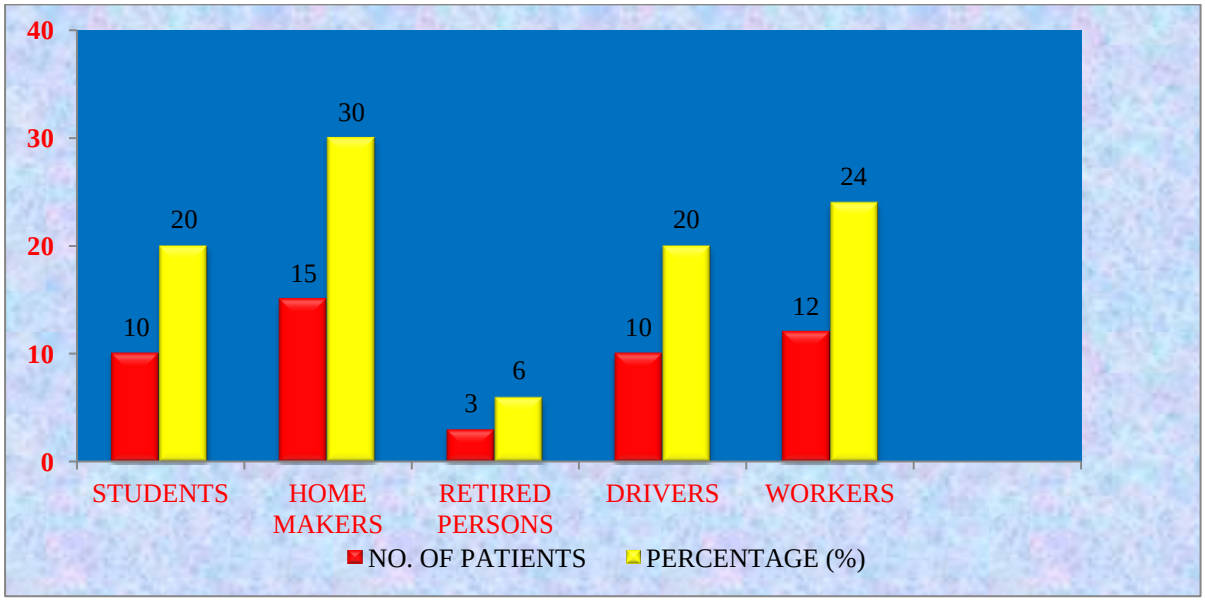
- 25 patients were poor.
- 10 patients were lower-middle.
- 10 patients were upper middle.
- 5 patients were rich.

Table.10

OCCUPATIONAL STATUS

SL. NO	OCCUPATION	NO. OF PATIENTS	PERCENTAGE (%)
1	Students	10	20
2	Home makers	15	30
3	Retired persons	3	6
4	Drivers	10	20
5	Workers	12	24
TOTAL		50	100

OCCUPATIONAL STATUS



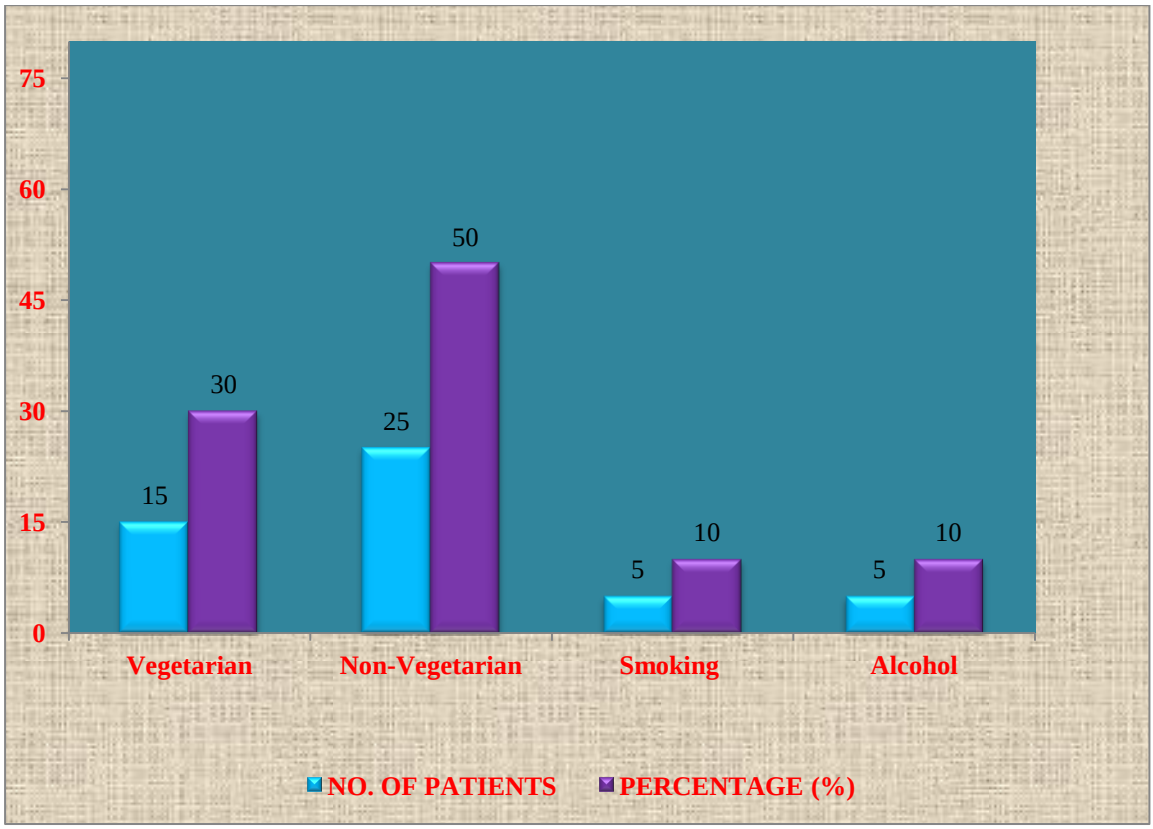
INFERENCE: Among 50 patients,

- 10 Patients were Students.
- 15 Patients were Home makers.
- 3 Patients were Retired persons.
- 10 Patients were Drivers.
- 12 Workers

Table.11

PERSONAL HABITS

SL. NO	PERSONAL HABITS	NO. OF PATIENTS	PERCENTAGE (%)
1	Vegetarian	15	30
2	Non-vegetarian	25	50
3	Smoking	5	10
4	Alcohol	5	10



5. RESULTS AND DISCUSSION:

Regarding the drug availability, the source of this study drug always meets the demands for the treatment for the whole year. It is a tree easy to collect. Here, various studies have been carried out in this study drug. The study includes literary collections, Pharmacognostic study, Physico and Phyto chemical analysis, toxicological study, pharmacological study, and clinical study. The drug has been selected for the treatment of *Pandu noi* in reference with *Gunapadam mooligai.*

Literary collections about the drug from various text books were done. It indicates the efficacy of the drug in the treatment of *Pandu noi.* Botanical aspect deals with the identification description, cultivation and ethno medicinal importance of the plant. *Gunapadam* aspect expressed that the drug possess good effect to treat Anaemia.

4.2.1. Pharmacognostic: Study showed Macroscopical and Microscopical

Macroscopical:

Colour	-	Greenish colour
Odour	-	Odourless
Taste	-	Sour

Microscopical:

Microscopic features of the leaflet:

The leaflet has less prominent midrib and thick lamina with smooth and even surfaces (Fig5.a). The midrib is flat in the adaxial side and slightly raised into shallow convex on the abaxial side (Fig5.b). The midrib is 220µm thick. The epidermal layer of the midrib is narrow the cells being cylindrical and thin walled. The palisade cells are transcurrent across the midrib vascular bundle, below the adaxiall epidermis.

The vascular bundle of the midrib is single, large and circular and measures 160µm in diameter. It consists of an are of 4 or 5short parallel rows of wide and thin walled xylem elements and a thick are of phloan cells to eated on the lower end of the xylem strand (Fig6.a). The entire vascular strand is encircled by thick axes of sclerenchyma cells on the adaxial and abaxial portious of the bundle (Fig 6.a,b)

Lamina (Fig 5.a):

The lamina is 160µm thick. It is dorsi ventral with distinction of adaxial and abaxial sides. The adaxial epidermis consists of wide and thick cells with thin cuticular layer(Fig5.c). The cells are20µm thick. The abaxial epidermis is comparatively thin;the cells are either cylindricalor broadly conical; the epidermis is stomatiferous. The mesophyll tissue is differentiated into adaxial zone of two layers of narrow cylindrical, compact cells.The abaxial zone micludes about eight layers of small lobed cells, loosely arranged cells with intracellular spaces(Fig5.c)

Leaf margin (Fig.6.c)

The marginal part of the lamina is thick and conical. It is 90µm thick. The epidermal cells along the marginal part are reduced in size; they are squares or rectangular thick walled with thick cuticle. A circular thick vascular bundle is located within the marginal part. The bundle consists of thick sclerenchymetous bundle sheath enclosing a few, thick walled xylem element and a few phloem elements.

PHARMACOGNOSTICAL FIGURE OF (*Tamarindus indica).*

Fig 5.a T.S of leaf through Lamina

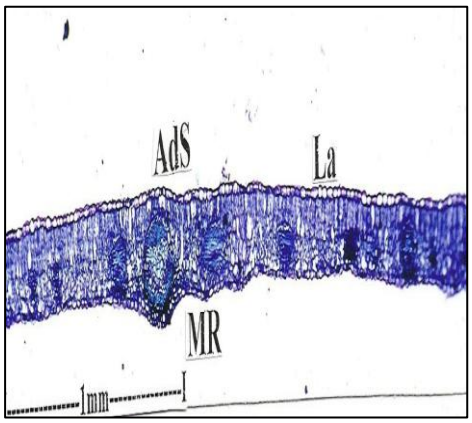


Fig 5.b T.S of Leaf through Lamina Midrib - enlarged

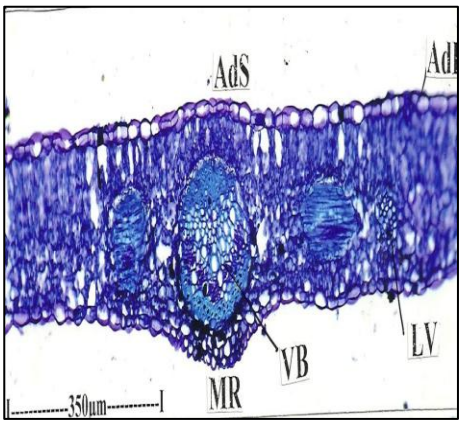


Figure 5.c Transverse Section of Lamina

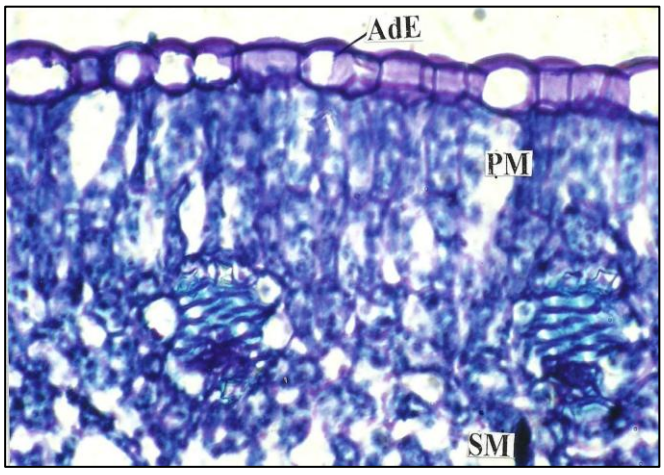


Figure 6.a Vascular Bundle of the Midrib – Enlarged

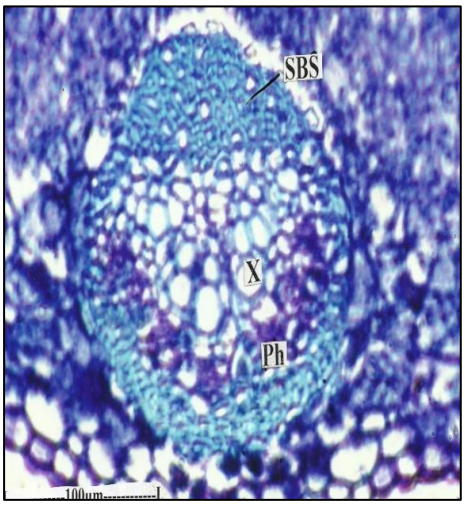


Figure.6.b.Vascular Bundle of the Midrib -As seen under polarized light

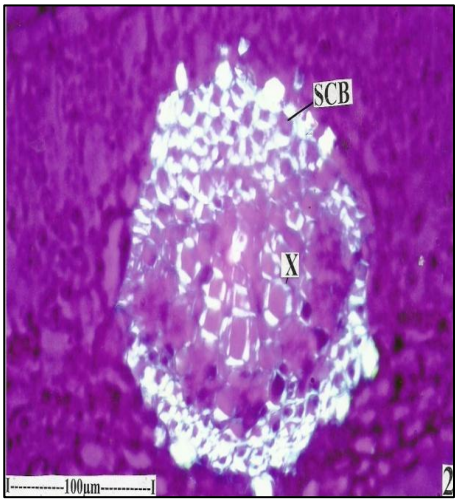


Figure 6.c Transverse section of leaf Through margin

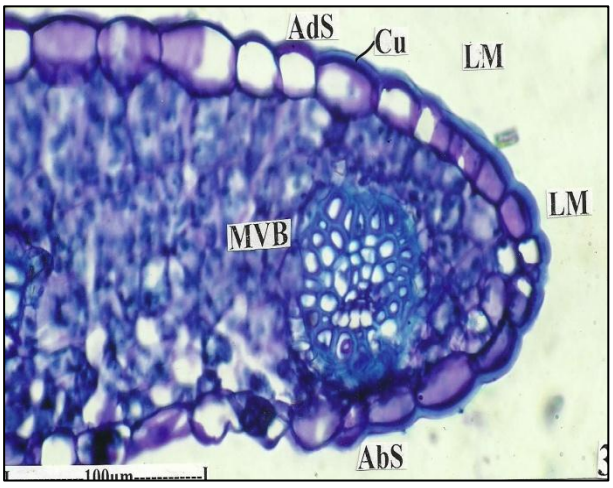


Table .12.

4.2.2. PHYSICO – CHEMICAL ANALYSIS:

S.No	Parameter	Mean Value
1	Loss on Drying at 105°C	4.5 %
2	Total Ash	6.75 %
3	Acid insoluble Ash	0.85 %
4	Water Soluble Extractive	28.9 %
5	Alcohol Soluble Extractive	22.2 %
6	Particle size	Completely passes through sieve no. 44
7	pH	3.0

4.2.3.QUALITATIVE PHYTOCHEMICAL TESTS

Table. 13

S.No	**Parameter**	**Mean value**
1	Alkaloids	- ve
2	Triterpenes	+ ve
3	Flavonoids	+ ve
4	Saponin	+ ve
5	Steroids	+ ve
6	Protein	+ ve
7	Glycoside	+ ve
8	Acid	+ ve

TLC:

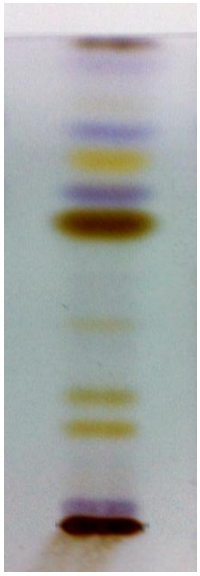


Figure. 7. TLC

After spray with visualizing agent Table .14.

Sl.No	After Dipping in Vanillin-Sulphuric acid	
	Rf value	**Colour of the spot**
1	0.04	Purple
2	0.21	Yellow
3	0.27	Yellow
4	0.42	Yellow
5	0.63	Yellowish brown
6	0.69	Purple
7	0.75	Yellow
8	0.81	Purple
9	0.95	Purple

Solvent system:

Toluene : Ethyl acetate (4:1.5).

TLC plate:

Aluminium plate precoated with silica gel $60F_{254}$ of 0.2 mm thickness (Merck).

Developing chamber:

Camag's twin trough chamber.

Visualizing reagent:

Vanillin-sulphuric acid reagent.

4.2.4. BIO – CHEMICAL ANALYSIS:

The Bio – Chemical analysis of *Puli ilai chooranam* showed following chemicals,

Reducing sugar, starch, protein, aminoacids, albumin, phosphate, sulphate, chloride, iron, calcium, magnesium, tannin.

4.3. TOXICOLOGICAL STUDY:

Acute Toxicity:

Table 15: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose mg/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	2000	+	+	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2	5000	+	+	-	+	+	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

4.4. PHARMACOLOGICAL STUDY:

The present study aimed to evaluate the effect of *Puli Ilai Chooranam* on the haemolytic anaemia induced by phenylhydrazine in albino rats. The *Puli Ilai Chooranam* appears safe for use since the tolerability of *Puli Ilai Chooranam* was greater than 5 g/kg. It has been demonstrated previously that intraperitoneal administration of phenylhydrazine decreased haemoglobin concentration, red blood cells number and haematocrit. In this study, PHZ altered the function of RBC by haemolysis characterized by decrease in RBC, Hb concentration, WBC and PCV. However, these parameters were restored to normal range after treatment with *Puli Ilai Chooranam* suggesting that have significant haematinic effect. The results of this study indicated that the *Puli Ilai Chooranam* increased significantly the concentration of haemoglobin, red blood cell count, white blood cell count and the packed cell volume mainly one week after of treatment.

Iron is essential for cellular metabolism, viability, and growth regulation. On the other hand, free iron is pro-oxidant and can, therefore, damage cells owing to the attack of reactive oxygen species and lipid peroxidation products on bioactive macromolecules, such as proteins and nucleic acids. Anaemia is a major cause of morbidity and mortality in malaria endemic areas of tropical regions of the world. Anaemia in malaria is caused by destruction of red blood cells in the body or the depression of red blood cell production in the bone marrow. As the red blood cells become less, the patient weakens. In severe cases the patient is unable to deal with basic life essential tasks and may die. There are various causes and types of anaemia, these include: sickle-cell anaemia, iron deficiency anaemia, vitamin B12 anaemia, drug induced anaemia as side effect of drug therapy.

The *Puli Ilai Chooranam* had a positive effect on the haemopoietic system of the test rats. It significantly increased the red cell mass, haematocrit, haemoglobin concentration and total white cell count while decreasing lymphocyte and monocyte counts. In the treatment of diseases like malaria and anaemia, the beneficial effects of analyzed drug materials especially from plants are mainly attributed to the presence of constituents like alkaloids, saponin, terpenoids, anthraquinones, essential oils, flavonoids, tannins, etc.

The efficacy of these constituents could also be influenced by inorganic components known as the trace elements. Micronutrient deficiencies and infectious

disease often coexist and show complex interactions leading to mutually reinforced detrimental clinical effects especially in underprivileged people of developing countries, particularly in rural regions. Several micronutrients such as trace elements (Zn, Fe, Se) modulate immune function and influence the susceptibility of the host to infection. Some trace elements have been found beneficial in the control of anaemia under this condition. The increase in the blood indices was progressive giving the highest effect on the second week of *Puli Ilai Chooranam* treatment may be more beneficial in the anaemic conditions.

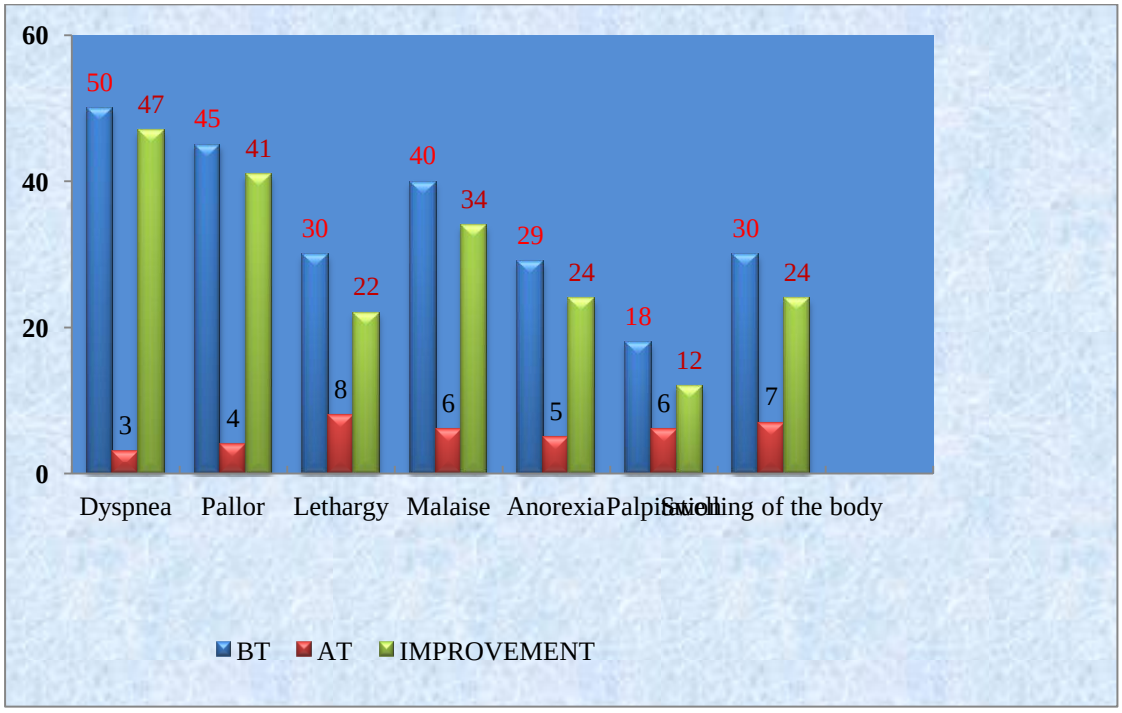
Under normal condition, the body can generate new RBCs to replace the lost red cells; this will take much longer time as shown in this study. The recovery time of two weeks for untreated rats has earlier been reported when rats were bled 20% of their total blood volume to induce haemorrhagic anaemia. The increases in the haematological indices exhibited by *Puli Ilai Chooranam* might be related to the vitamin and mineral contents of the *Puli Ilai Chooranam*. These constituents are well known haemopoietic factors that have direct influence on the production of blood in the bone marrow.

4.5. CLINICAL ASSESMENT

Table .16. IMPROVEMENTS IN SIGNS AND SYMPTOMS

SL. NO	SIGNS AND SYMPTOMS	NO. OF PATIENTS			
		BT	AT	IMP.	IMPROVEMENT (%)
1	Dyspnoea	50	3	47	94
2	Pallor	45	4	41	91
3	Lethargy	30	8	22	73
4	Malaise	40	6	34	85
5	Anorexia	29	5	24	82
6	Palpitation	18	6	12	66
7	Swelling of the body	30	7	24	80

IMPROVEMENTS IN SIGNS AND SYMPTOMS



INFERENCE:

Among 50 patients,

- 47 out of 50 patients were relieved from Dyspnoea.
- 41 out of 45 patients were relieved from Pallor.
- 22 out of 30 patients were relieved from Lethargy.
- 34 out of 40 patients were relieved from Malaise.
- 24 out of 29 patients were relieved from Anorexia.
- 12 out of 18 patients were relieved from Palpitation.
- 24 out of 30 patients were relieved from Swelling of the Body.

Gradation result

Grading of Anaemia:

Grade 0 - With in normal level

Grade 1 - Lower normal limit 10g/dl

Grade 2 - 8 – 10 g/dl

Grade 3 - 6.5 – 8 g/dl

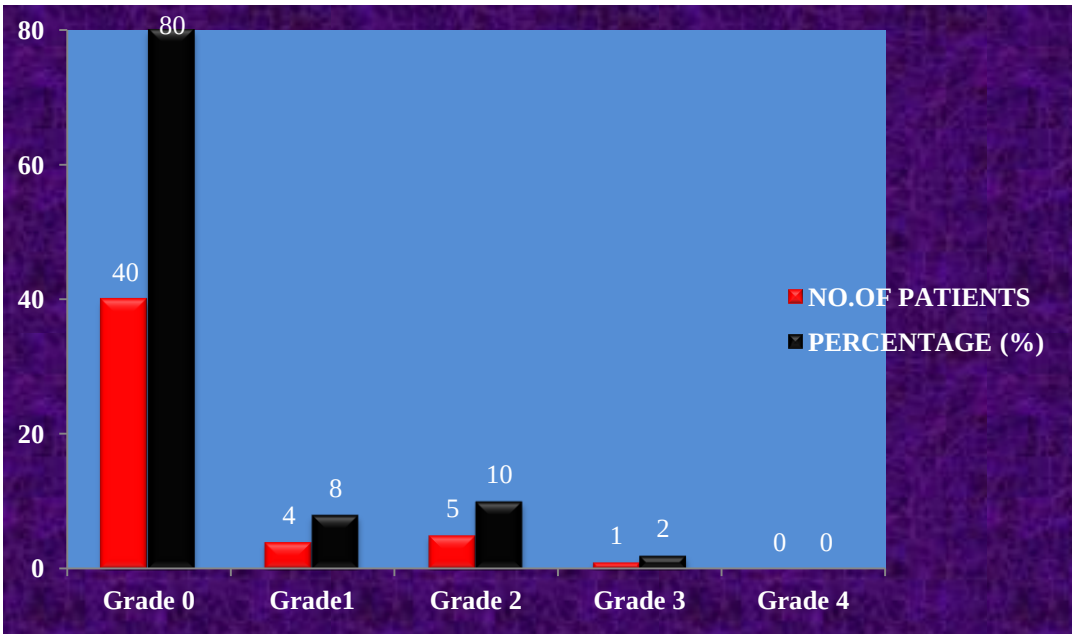
Grade 4 - < 6.5 g/dl

Grade 5 - Death.

Table, 17. Grading of Anaemia

S.No	GRADE	NO OF PATIENTS	PERCENTAGE(%)
1	Grade 0	40	80
2	Grade 1	4	8
3	Grade 2	5	10
4	Grade 3	1	2
5	Grade 4	0	0
6	Grade 5	0	0

GRADATION RESULT:



Clinical study:

50 patients of both sexes were selected .Among the 50 patients, 40 patients were treated as out- patients in the Post graduate department of Gunapadam, Govt.Siddha medical college hospital and Govt. Arignar Anna hospital, Arumbakkam, Chennai- 106. 10 patients were treated as in - patients.The patients were observed regularly.

The trial drug *Puli ilai chooranam* was given to the patients at the dose of 1 gm twice a day with hot water before meals. On administration of *Puli ilai chooranam* 1 gm twice a day for 7 weeks should significant Haematinic activity. Honey which was used as vehicle also has iron containing property as per classical siddha literature.

Among 50 patients, 47 out of 50 patients were relieved from Dyspnoea. 41 out of 45 patients were relieved from Pallor . 22 out of 30 patients were relieved from Lethargy. 34 out of 40 patients were relieved from Malaise. 24 out of 29 patients were relieved from Anorexia. 12 out of 18 patients were relieved from Palpitation. 24 out of 30 patients were relieved from Swelling of the body. The results revealed that the drug possess 80% Grade 0, 8% Grade 1, 10% Grade 2, 2% Grade 3.

STATISTICAL ANALYSIS

DESCRIPTIVE STATISTICAL FOR IMPROVEMENT OF SIGNS & SYMPTOMS IN *"ANAEMIA"*

PAIRED "t" TEST RESULT:

Table.18."**p" value & statistical significance:**

Treatment	Signs & symptoms	Mean	S.D	S.E.M
Before treatment	7	34.57	10.98	4.15
After treatment	7	30.00	13.04	5.32

From the table we calculated the descriptive statistic like Mean, S.D & S.E.M of Mean for the improvement score before and after treatment.

"t" Table: 19

t-Table	S.D	"t" Value	"p" Value
Pre vs Post	0.715	7.46	0.0007

The two-tailed P value is less than 0.0007
By conventional criteria, this difference is considered to be extremely statistically significant.

RESULT AND DISCUSSION:

Improvements of signs and symptoms by statistical analysis shows the two tailed "p" value equals 0.0007, by conventional criteria, this difference is considered to be extremely statistically significant. From the above results $p < 0.05$, it shows the improvement in the subjective parameters produced by *Puli ilai chooranam* statistically significant.

6. CONCLUSION

The trial drug of "***Puli ilai Chooranam***" has been selected and its efficacy was analyzed in the treatment of Anaemia.

The drug is simply available and preparation is very easy.

The trial medicine is cost effective.

The drug shows good Haemataenic activity.

No adverse effects were produced during the entire clinical trial.

From the above observation, I conclude that the drug **"*Puli ilai chooranam*" (*Tamarindus indica*)** gives a new hope in the field of Anaemia Treatment.

7. SUMMARY

The tree ***Tamarindus indica*** leaves were collected from *Vadalur, Cudalore* (Dt) and purified then powered and stored. This drug was subjected to various studies.

Puli ilai Chooranam was selected for this study to estimate the Haemataenic activity, and proved its efficacy and safety in Anaemia disease.

I was collected the information about the drug, various text books, Literature was referred. From them, i came to an idea about the drug and its worth on Anaemia.

A brief description about botanical aspect of the herb ***Puli ilai*** and its identifying characters and Phyto chemical data's were given.

The pharmacological analysis showed that the drug has got significant Haemataenic activity.

In clinical study, the drug has showed marked improvement in 80% of cases.

The patients were responding well from the beginning of the treatment and no adverse effects were reported.

This present study suggests that ***Puli ilai Chooranam*** has the remarkable medicinal value against the disease Anaemia without any adverse effect.

1. INTRODUCTION

In the world more than 20,000 traditional forms of medical systems are available. Some of them are popular as well as mostly used by the people for their basic and primary health care even today.

Siddha Medical system or Tamil Medical system is one of the ancient systems of medicine, which is being in existence. It has got valid scientific, significant, the most respectable and of high order of medical system. It is being practiced extensively in Southern India, Srilanka, Malaysia and Singapore, where the Tamil civilization had taken its roots.

The typical characteristic features of the *Siddha Materia Medica* are utilization of metal and mineral based preparations to a greater extent in comparison with other traditional systems of medicine. The *Siddha*rs were the pioneer in the use of metals and minerals in the treatment of diseases.

It has been noted that metallic or mineral medicines can help tremendously in patients with chronic or degenerative diseases and have been tried by all other medical systems and that too after treating primarily with herbal medicines. The same thing is conveyed by Siddhars before many years. They laid down the line of treatment to give first preference to herbal medicines and then to go on with herbo-mineral and then to metallic preparations. This is revealed in the following verse as

"வேர்பாரு தழைபாரு மிஞ்சினக்கால்

மெல்ல மெல்ல பற்ப செந்தூரம் பாரே"

சித்த மருத்துவாங்க சுருக்கம் பக்கம் 473

Treating the minerals with herbal juices may lead to reduction (trituration) in particulate size even up to Nano levels (less than 100 nm) which enable increased potency. These drugs are known to be effective even in low concentration. This reduction of particle size of *Parpam, Chendooram* and *Chunnam* are intimately connected with the size of today's so called "**Nano particles**". The prevailing "Nano medicine system" would be the basic tool for developing new drugs, which is prime dream for every scientist. But this secret was already established and documented by

our *Siddhars* prior to many generations. This is considered to be one of the gems in the field of *Siddha* Medicines. This shows their vast knowledge about the medicine and hence for the reason, they are considered to be immortals and the *Siddha* system of medicine is the perfect medical science of the past, present and future. One of the unique and vital categories in *Siddha* System of Medicine is *Parpam.*

Basically, *Parpam* is considered as one of the alkaline medicines which contains mainly calcium and is used for various medical purposes. Scientists have found that most diseases thrive in an acid environment, but cannot survive in an alkaline environment. It is a proven scientific fact that the bodily fluids (e.g., blood, spinal fluid, saliva, etc.) of a healthy human body are alkaline (with a high pH reading), whereas the bodily fluids of a person who is sick are acidic (with a low pH reading).

Charam are often considered the medicine cabinets of the 21st century. But the focus on The unique medical properties of *Charam* were recognized by *Siddhars* in pre-historic period itself.

Purified *charam* are grinded into powder and can be used to cure diseases of liver. *Charam* is given as a protective amulet.

Liver is the largest internal organ in the body and has more functions than any other human organ. It plays a central role in many essential physiological process, including glucose homeostasis, plasma protein synthesis, lipid and lipoprotein synthesis, bile acid synthesis and secretion and vitamin storage (B12, A, D, E and K). It is also the principal organ related to metabolism and excretion of a wide variety of environmental pollutants and also therapeutic agents.

As per the global estimates, there are about 18, 00,000 deaths every year due to liver cirrhosis, mainly caused by hepatitis. Although viruses remain the main cause of liver diseases, the liver lesions arising due to imbalance of enzymatic levels which result from xenobiotics, excessive drug therapy, environmental pollution and alcoholic intoxication are among the secondary reasons (Handa SS, 1991).

Based on the above facts, the liver disorders are one of the leading serious world health problems prevailing today. Despite its frequent occurrence, high

morbidity and high mortality, its medical management is currently not satisfactory and inadequate. No therapy has successfully prevented the progression of hepatic diseases. In the absence of reliable hepato-protective drugs in the market, herbal, mineral and animal derived preparations play a vital role in the management of various liver disorders. Numerous medicinal plants and their compound formulations are used for liver disorders in *Siddha* medical practices and other traditional systems of medicine in India. However, there is increasing problem of liver disorders which demands for more precise, safe and effective treatments for the same is the need of the hour.

So, the search for effective hepato-protective drug still continues...

With the statements made above, *charam*, with the preparation, can be considered as one of the primary *Siddha* medicines which could fetch effective results for the cure of liver disorders. So, the author is interested to evaluate the *Chara parpam***,** a unique herbo- mineral drug preparation for its hepato-protective activity.

This *Siddha* drug "C*hara parpam"* is yet remained unexplored for its exact chemical, pharmacological and clinical values in terms of scientific research. To fill these scientific lacunae, the present work was undertaken to evaluate the chemical, pharmacological and clinical efficacy of ***Chara Parpam***.

2. AIM AND OBJECTIVES

Liver diseases are among the important health disorders affecting millions of people worldwide. Since 300 million people (about 5% of the world population) suffer from various hepatic disorders which are the main cause of cirrhosis and liver cancer, particularly in developing countries like India.

Apart from viral origin, common liver disorders are mainly induced by hepatotoxins, which includes alcoholic hepatitis and drug induced hepatitis.

Hepatotoxicity is a growing concern of today's modern society. The increasing incidence of alcoholism, cigarette smoking, fast foods and other xenobiotics have contributed to the morbidity and mortality due to liver disease. Jaundice and hepatitis are two major hepatic disorders that accounts for a high death rate. Especially liver disorder is one of the common causes of death between the age of 25 and 44.

Modern medicine has little to offer for alleviation of hepatic diseases and even most of them are based on herbal preparations only. But there are not much drugs available for the treatment of liver disorder. (Karan et al, 1999, Chaterrjee 2000). In absence of reliable liver protective drugs in modern medicine, there exists a challenge for pharmaceutical scientists to explore the potential of hepatoprotective activity using natural sources. In this situation traditional medical systems play an important role in the treatment of various hepatic disorders.

Among the various sources for the development of potent hepatoprotective drugs, compounds from living organisms especially from marine origin are of particular significance. In fact, one coral reef ecologist Andrew W.Bruckner says that, we are 300 to 400 times more likely to find new drugs in the ocean than on land.

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- The present study was focused to evaluate the hepatoprotective potentials of *Chara parpam* in experimental animals with CCl_4 induced hepato toxicity.

- It was also carried out to determine the clinical efficacy of *Chara parpam* in patients with Jaundice (hyperbilirubinaemia) due to hepatic pathology.

2.1. OBJECTIVE:

- This scientific study on Chara parpam was carried out in the following stages.
- To Collect the literature evidences related to the trail drug
- To Get the proper authentication of Raw drugs
- Preparation of the trial drug, according to the text in a classic method.
- Physico-chemical, Chemical Analysis for the trial drug to identify the active mechanism
- Toxicological studies to prove the safety of the drug.
- Pharmacological study to evaluate the hepatoprotective efficacy of the drug
- Clinical studies Evaluating the therapeutic efficacy of *Chara parpam* through open clinical trial on *KAMALAI* patients

3. REVIEW OF LITERATUE

3.1. Gunapadam aspect of the drug:

3.1.1. நவாச்சாரம்

வேறுபெயர்:

இஷ்டிகை

சுல்லிகை

சூளிகை

படு

அசுரன்

நவாச்சாரத்தின் தோற்றம்:

- கற்சூளைகளில் இது கிடைக்கும்
- இது ஒட்டகம் முதலிய மிருகங்களின் சாணச்சாம்பலுடன் நிலக்கரியையும் உப்பையுங் கூட்டிப் பதங்கித்து எடுக்கப்படுகின்றன.

பண்பு:

கடைகளில் கிடைக்கும் சரக்கு பார்வைக்கு கட்டியாயும் வாசனையின்றியும் நார்நாராயும் தூள்செய்ய கடினமாயும் இருக்கும். நீரிலும் சாராயத்திலும் கரையக்கூடியது. நவாச்சாரம் செயற்கை உப்பு வகையை சார்ந்தது.

சுாரத்தின் வைப்பு

யானை, குதிரை, கழுதை, ஆடு, மாடு, ஒட்டகம், பன்றி, மனிதன் இவற்றின் சிறுநீர் சமஅளவாக சேர்த்து அதற்கு 1/8 பங்கு சூடனும், சீனமும்கூட்டி பாண்டத்தில் எரித்து கட்டியான பின் பொடிசெய்து ஒரு வாய்குவிந்த பாண்டத்தில் 1/2பங்குவரை போட்டு சில்லிட்டு சீலை செய்து அடுப்பேற்றி 5 நாள் கமலாக்கினியாக எரித்து புடம் போட்டு 5 நாள் உடைத்து பார்த்தால் சாரம் பலகையாகும்.

நவச்சாரத்தின் சத்துரு மித்திரு:

சத்துரு	
கல்லுப்பு	சுக்கான்
இந்துப்பு	காரீயம்
படிகம்	கடல்நுரை
வளையலுப்பு	அப்பிரகம்
இரும்பு	சவுடு
காந்தம்	கிளிஞ்சில்

மித்துரு

தாளகம்	சிலை	கல்நார்	நாகம்
வெடியுப்பு	வீரம்	நிமிளை	செம்பு
இலிங்கம்	வெங்காயம்		

நிறம்: அழுக்கு படிந்த வெண்மை (அ) கபில நிறமுடையது.

சுவை: கசப்பு, புளிப்பு மூத்திர வெகுட்டல் உடையது.

சுத்திமுறை:

நவாச்சாரத்தை வெந்நீரில் கரைத்து சூடாயிருக்கும்போது வடிகட்டி குளிர் ஆறினபின் வாயகன்ற பாத்திரத்திலிட்டு வெய்யிலில் வைக்க உப்பு உறையும். அதை புட்டியில் அடைத்து பத்திரப்படுத்தவும்.

செய்கை:

குறைந்த அளவில் நாட்படக் கொடுத்தால் உடல்தேற்றியாகும். அதிகஅளவில் கொடுக்க வெப்பமுண்டாக்கியாகும்.

கோழையகற்றி, வியர்வைபெருக்கி, சிறுநீர்பெருக்கி, விரணமுண்டாக்கி, பித்தமகற்றி.

இது முக்கியமாக நிணநரம்புகள், மாமிசக்கிரந்திகள் மீது தன் வேகத்தைச் செலுத்தும்.

பொதுக்குணம்:

“ குன்மம் குடற்சூலை கொல்லும் மகோதரத்தை
வன்மையுறு கல்லடைப்பை மாற்றுங்காண் - சன்மக்
கவிச்சுமுத் தோடங் கனவாத நீக்கும்
நவச்சார மாதே நவில்.”

நவச்சாரம் வயிற்றுவலி குடலில்குத்தல், பெருவயிறு, கல்லடைப்பு, சருமத்தில் புலால்வாசம், திரிதோடம், கனவாயு இவைகளை நீக்கும்.

உப்புசம், கல்லீரல்வீக்கம், பிலீகநோய், நீர்க்கோவை, இரத்தகாசம், முகச்சந்நி, சூரியாவர்த்தவாதம், சூதகக்கட்டு, கக்கிருமல், முறைக்காய்ச்சல், விடாக்காய்ச்சல் இவைகட்கும் உபயோகிக்கலாம்.

பயன்கள்:

மஞ்சள்காமாலை ஊதுகாமாலை கல்லீரல்வீக்கம் கல்லீரலில் உண்டாகும் கட்டி முதலிய நோய்களுக்கு நவாச்சாரத்தை நீர்முள்ளிக்குடிநீரில் கொடுக்க சிறுநீரைப் பெருக்கிப் பிணியை நீக்கும்.

சேருமருந்துகள்:

1.நவாச்சாரக்குழம்பு:

1 பலம்(35கிராம்) நவாச்சாரத்தைக் கல்வத்திலிட்டுக் கொடிக்கள்ளிப்பால் விட்டு நன்றாய் மைபோலரைத்துச் சிமிழில் பதனம் பண்ணிக் கொள்ளவும்.

அளவு : குன்றியளவு (130மி.கி)

தீரும்நோய்கள்: மலக்கட்டு சலக்கட்டு பெருவயிறு வாயுகட்டி நீராம்பல்.

2.**இலவணக் குழம்பு: (**அனுபோக வைத்திய நவநீதம் பாகம் 3)

கறியுப்பு 5 பலம்

அப்பளகாரம், வெண்காரம், அனைத்தும்

நவச்சாரம், கற்பூரம், வகைக்கு 1 / 2 பலம்

வெடியுப்பு, கடல்நுரை

சீனாக்காரம், பெருங்காயம்,

இந்துப்பு, சவுக்காரம்,

பசுவின் நீர் - 2 படி

பனைவெல்லம் - 10 பலம்

மேற்படி வெல்லத்தைக் கரைத்து உப்புகளையும் மற்ற சரக்குகளையும் நுண்மையாகப் பொடித்து அதிற்போட்டு அடுப்பேற்றி சிறு தீயாக எடுத்து மெழுகு பதத்தில் பீங்கான் பாத்திரத்தில் வைத்துக்கொள்ளவும்.

அளவு: 1 முதல் 11/2 வராகன் எடை காலை மாலை இரண்டு வேளையும் உபயோகிக்கலாம்

தீரும் நோய்கள்:

மகோகதரம், பெருவயிறு, பாண்டுநோய், **காமாலை நோய்கள்**, கவுசை கட்டிகள், பீலிகைக் கட்டிகள், அகட்டுவாயு, பாண்டு முதலிய நோய்கள் தீரும்.

3.நவச்சாராதிக்குழம்பு

நவச்சாரம், இந்துப்பு, வெடியுப்பு, ஓமத்தூள், சவுக்காரம், காந்தத்தூள், கடுகுத்தூள் தூய்மை செய்த நேர்வாளப்பருப்பு, மஞ்சள், கடுக்காய்த்தோல் சூரணம், இவை வகைக்கு 1 பலம் (35பலம்) 5 பலம்(175கிராம்) மஞ்சள் கடுக்காய் தோலை ஒன்றைப்படி தண்ணீரில் போட்டு அரைப்படியாகச் சுண்டவைத்து திறந்து வடிகட்டின குடிநீர் செல்லத்தக்க அளவு.

செய்முறை: ஆரம்பத்தில் வாளத்தைக் கல்வத்திலிட்டுத் தூள் செய்து மற்றவைகளையும் முறையே ஒவ்வொன்றாக சேர்த்தரைத்து, முடிவாக எல்லாவற்றையும் ஒன்றாகச் சேர்த்து மேற்படி குடிநீரைச் சிறுகச் சிறுக வார்த்து 4 சாமம் அரைத்து மெழுகு பதத்தில் பீங்கான் சிமிழில் பத்திரப்படுத்தவும். அளவு: 2 – 3 குன்றிஎடை.

துணைமருந்து: சிற்றாமணக்கு எண்ணெய், இஞ்சிச்சாறு, சுக்குக்குடிநீர், சோம்புகுடிநீர். **தீரும்நோய்கள்:** நீராமை, **பெருவயிறு,** கவிசை, வயிற்றுக்கட்டி, **பித்தகாமாலை,** எட்டுவகைபாண்டு.

3.2.2.வெடியுப்பு

POTASSH NITRAS; POTASSIUM NITRATE.SALT PETRE; NITRATE OF POTASH.

வேறுபெயர்:

பொட்டிலுப்பு

இணங்கன்

படைராசன்

பூமி கூர்மை

நவச்சார மித்ரு

வைப்பு முறை:

ஓரடி கனத்த மட்பாண்டத்தில் உப்பு உதர்ந்த மண்ணைக் கொட்டி நீர்விட்டுக் கலக்கிப் பிறகு குருது கட்டி தமரிட்டு வைக்கோல் சொருகி மேற்படி நீரை விட்டுத் தெளிவெடுத்து அதனை காய்ச்ச உப்பாகும்.

இவ்வுப்பு 1 க்கு நீர் நான்குபங்கு விட்டுக்காய்ச்சும்போது முப்பத்திற்கு 1 பங்கு புளித்த மோர் பழச்சாறு இவற்றை விட்டு காய்ச்சி உப்பெடுக்கவும்.இப்படி 4 அல்லது 5 முறை காய்ச்சி எடுக்க உப்பு கம்பி கம்பியாய் நிற்கும். இது வாதத்திற்கு வேர்.காய் இலை பூ என்பர்.

சுத்திமுறை:

வெடியுப்பு - 1 பங்கு

தண்ணீர் - 4 பங்கு விட்டு அடுப்பேற்றி சிறு தீயால் எரித்துக் கொதிகிளம்பும்போது,1 வீசை உப்புக்கு 4 கோழி முட்டை வெண்கருவைச் சேர்க்க வேண்டும். மேலே அழுக்குத் திரளும். அதனை அகப்பையால் வழித்து நீக்கி, உறையும் பதத்தில் மறுசட்டியில் சீலைகட்டிஅதில் வடித்துக் காற்றில்லா விடத்தில் வைத்து,மறுநாள் நீரை வடித்துவிட்டு,சூரியவொளியில் உப்பை உலர்த்தவும். இவ்வாறு 7 முறை செய்யச் சுத்தியாம்.

வேறுமுறை:

வெடியுப்பு 1 பங்கு கடல்நீர் அல்லது நீர் 2 பங்கு எடுத்து உப்பை நுண்மையாய்ப் பொடிசெய்து நீரில் கலந்துவைக்க நீரில் கலந்துபோம். தெளிவெடுத்து வெண்மையான இருப்புப் பாண்டத்தில் விட்டுக்காய்ச்சி உறையும் பதத்தில் வேறு ஒரு செப்புப்பாண்டத்தில் கொட்டி குளிர்ந்த இடத்தில் ஆறவைக்க உப்பாகும். இதை எடுத்து இதற்கு 2 பங்கு நீர் விட்டு மேற்படியாகவே காய்ச்சி உப்பாக்கவும். இப்படி மொத்தத்தில் 5 முதல் 7முறை செய்ய சுத்தியாம்.

சுத்தி செய்த உப்பு கம்பிகளாயும் வெண்மையாயும் நாக்கிலிட்டால் குளிர்ச்சியாயும் இருக்கும். நெருப்பிலிட பொரியும்.

செய்கை:

உட்பிரயோகம்:

குளிர்ச்சியுண்டாக்கி

வியர்வை பெருக்கி

சிறுநீர்பெருக்கி

வெளிப்பிரயோகம்:

குளிர்ச்சியுண்டாக்கி

பொதுகுணம்:

மல்லாரு மட்டகுன்ம மாதருத ரக்கட்டி

கல்லா மதைப்புநீர்க் கட்டருக – லெல்லாமே

கம்பிகம்பி யென்றுங் கருவுண்டா மங்கிநின்ற

கம்பிகம்பி யென்றுரைக்குங்கால.

சூதகவாயுவொடு சோணிதத்தின் வாதமும்போம்

வுாதவலி குன்மமிவை மாறுங்கான் - மீதாங்

கோடிய வயிறிழியுங் கோழைகப மேகும்

வெடியுப்புத் தன்னை விளம்பு.

குன்மம் கருப்பாசயக்கட்டி சோபை மூத்திரகிரிச்சரம் நீர்சுருக்கு சூதிகாவாதம் வாதசோணிதம் பெருவயிறு ஈளை கபதோடம் இவை ஒழியும்.பேரிளம் பெண் பருவங் கடந்த மாதர்கட்கும் கருப்பம் உண்டாகும்.

இதனால் சுரம் வீக்கம் கீல்வாதம் இரத்தபித்தம் பிரமேகம் கண்ணோய் தொண்டை விரணம் சுவாசகாசம் முதலியனவும் நீங்கும்.

3.1.3.ஆடாதோடை:

வேறுபெயர்கள்: ஆடாதோடை , வாசை

பயன்படும் உறுப்புகள்: இலை, பூ, பட்டை, வேர்.

சுவை - கைப்பு

தன்மை - வெப்பம்

பிரிவு - கார்ப்பு

பொதுகுணம்:

"ஆடாதோ டைக்கிரத்த பித்தமறுங் காச
மானந்த வாயுடன் மேலிளைப்பு மேகம்
கூடாகும் தாபசுரம் பித்தகப வாத
சுரரோகஞ் சந்நிபா தஞ்சூலை குட்டம்
ஓடாவோ வாந்தி விக்கல் மூலரோகம்
ஒளிதோடம் உட்கனலும் ஒழியுந் தானே
வாடாது மனக்கிளர்ச்சி யாகுமிதன் பெருமை
வகுத்துரைத்தார் முன்னோர்கள் வாழ்ந்திடயா வருமே."

குருதியழல், இருமல், மேலிளைப்பு, வாந்தி, விக்கல், சூலை, ஈவை போகும்.

3.1.4. துணைமருந்து:

சீரகம்:

வேறுபெயர்:

அசை, சீரி, நற்சீரி, மேத்தியம், பித்தநாசினி, போசனகுடோரி.

பயன்படும் உறுப்பு: விதை

சுவை: கார்ப்பு இனிப்பு **தன்மை**: தட்பம் **பிரிவு:** இனிப்பு

செய்கை:

அகட்டுவாய்வகற்றி
வெப்பமுண்டாக்கி
பசித்தீத்தூண்டி
துவர்ப்பி

பொதுகுணம்:

"பித்தமெனு மந்திரியைப் பின்னப் படுத்தியவன்

சத்துருவை யுந்துறந்து சாதித்து – மத்தனெனும்

ராசனையு மீவென்று நண்பைப் பலப்படுத்தி

போசனகு டாரிசெயும் போர்" **- தேரன் வெண்பா.**

பயன்கள்:

சீரகத்தை கையாந்தகரை சாற்றில் ஊறப்போட்டு எடுத்த பொடி 4 கிராம், எடை சர்க்கரை 2கி எடை, சுக்குப்பொடி 2கி எடை, இம்மூன்றையும் கலந்து தினமிருவேளையும் உட்கொள்ள **காமாலை** தீரும்.

சீரகம் 34கி உப்பு தேவையான அளவு சேர்த்தரைத்து, நெய்விட்டுத் தாளித்து தேன் அல்லது சர்க்கரையுடன் கலந்து **தீ குற்றத்தால்** பிறந்தநோய்களுக்கு கொடுக்கலாம்.

3.2. CHEMICAL ASPECT:

3.2.1.Ammonium chloridum (or) Ammonium chloride.

Vernacular name:

Sans :	Sal Ammoniae
Arab:	Armina
Ben:	Navasagara, Nishadal
Hindi:	Navasadara
Guj.Mah&Kon :	Navasagar
Tamil:	*Navacharum*
Burm:	Lovas, Zarasa.

Source:

It is obtained by the combustion of excretions of various animals (or) of animal matters (or) by burning coals (or) common salt. It is a secondary product in the manufacture of coal gas. It is generally very impure in dirty white or brownish translucent cakes. It is manufactured from a kind of day found at karnal in the Punjab.

It is generally obtained in india from unburnt extremities of brick kilns in which manure of animals.

Characters:

Obtained in white granular crystals or transparent masses. It is readily soluble in water and is highly deliquescent. It has a saline disagreeable, nauseous and pungent taste. It can be purified and made into a powder by dissolving in hot water and evaporating to dryness and then bottling.

Action:

It is alternative, expectorant which cholagogue in small doses, in large doses purgative. It has a marked stimulating action on the mucuous membranes. Increasing their secretion also on the absorbent system and on gland structures.

Uses:

- It relieves hepatic congestion and modifies hepatic secretions.
- Useful in cases of hepatic abscess, chronic hepatic congestion and in dropsy connected with the liver and ovarian diseases.
- In cirrhosis and in jaundice from catarrh of the bile ducts.
- For hepatitis, Sal – ammoniac 8 – 15 grains, mixed with 105 grains of absinthium (warm wood), rubbed well in a mortar with a little water and given in a single dose will give relief.
- It is valuable combined with liquid extract of glycyrrhiza or group of country liquorice and with a few grains of powdered cinnamon, in cases of whooping cough.
- In Amenorrohea, Dysmenorrhoea, gleet, leucorrhoea, chronic dysentery, and other similar chronic discharge from lungs stomach and other internal organs.
- In hysteria, nervousness, jaundice, and other liver complains and gastric catarrh, doses of 10- 20 grains three times daily are beneficial.
- It is often prescribed as a stimulating expectorant in chronic bronchitis and in pneumonia in the stage of resolution[chopra]
- In various form of neuralgia, in chronic liver diseases, organic or functional in rheumatic affections of the face.
- Externally its solution combined with nitre is a nice cooling and stimulating application to the head in headache.

3.2.2.VEDIYUPPU

[Potassium nitrate (KNO_3)]

Vernacular name:

Arab	-	*Abkar*
Burma	-	*Yand Zeing*
Canada	-	*Patluppu*
English	-	Potassium Nitrate (KNO3)
Hindi	-	*Shora*
Maharashtra	-	*Shora – Mithra*
Malay	-	*Sundawa*
Malayalam	-	*Veti – Uppu*
Sanskrit	-	*Saind larea*
Tamil	-	***Pottil – Uppu***
Telugu	-	*Pattu – Uppu*

Availability of *Vediyuppu:*

It occurs extensively in Bengal, Punjab and Northern India naturally as an efforesence on the soil. But the drug available now in the market is unnatural,

For medicinal use:

It act on the vascular system and thus reduces the frequency of the pulse. Given in the solid form or in concentrated solution it acts as irritant.

It is useful also in the early stages of dropsy, in cases of smallpox, measles, influenza, catarrh, gonorrhea, acute rheumatism, bleeding from the lungs, stomach, uterus or other internal organs attended by fever.

A compound preparation known as Laghu Sankha Dravakam, which smells strongly of nitrous fumes and which is made of country nitre 6 palams, yavakshara, Ammonium chloride, Borax and vit salt 2 palams each and Gandhaka vediuppu(a nitre variety), soda carbonas, ferrous sulphate, copper sulphate and black salt (suvarchala – uppu) 1 palam each, all powdered and distilled, is recommended for the relief of all liver complaints, by Vaidyas.

3.2.3. Justicia adatoda

Vernacular name:

Sanskrit	- *Sinhaparn, Vasaka*
Hindi	- *Adosa, Arusha*
Benghal	- *Adulsa*
Persia	- *Basnsa*
Tamil	- *Adhatodai*
Malayalam	- *Ataloetakam*
Gujarathi	- *Aduraspee*
Punjab	- *Bhekkar*

Habitat: The plant grows in most parts of India especially in the lower Himalayan ranges.

Parts Used: Leaves, roots, flowers and bark.

Constituents: Fat, Resin, Vasicine, Adatodic acid, Gum, Colouringmatter, Salts.

Properties:

The base visicine or vasicine, is monobasic and occurs as white needle – shaped crystals and has a melting point of 190 – 191: or 182 C. it is easily soluble in alcohol, is slightly soluble in cold water but more so in hot water with an alkaline reaction.

Visicine hydrochloride occurs in light, cream – coloured crystals, has a melting point of 180c and is very soluble salt. The molecular weight of viscine was determined and found to be 188 which agree with the empirical formula C11 H12 N2O found by analysis.

Action:

Expectorant,
Diuretic
Antispasmodic
Alterative.

3.3. SIDDHA ASPECTS OF THE DISEASE:

3.3.1. மஞ்சள்நோய் (காமாலை):

வேறுபெயர்கள்:

காமாலை,

பித்து நோய்,

காமிலா

காமலா,

இயல்பு:

சிறுநீர், கண், நா, உடல் யாவும் மஞ்சள் நிறத்தைப் பெறும் நோயாம்.

நோய் வரும்வழி:

பித்தத்தை பெருக்கக்கூடிய செயல், அளவுக்கு அதிக உணவையும் கொள்ளின், கேடடைந்த குற்றத்தின் அளவாய் குருதிகெட்டு, பித்தநீரை குருதியிலும். உடல் உறுப்புகளாகிய தசை, தோல், கண், நாக்கு இவற்றில் தங்கசெய்து நோயை உண்டாக்கும்.

முற்குறிகுணங்கள்:

“பருகவே உள்ளங்கா லுள்ளங் கைகள்
பகர்முகங்கண் ணுடம்புமிக வெளுப்பு காணுங்
கருகவே கால்கைக ளோய்ச்ச லாகுங்
கனமாக நடுக்கியே இளைப்புண் டாக்குஞ்
சுருகவே மலந்தானும் வறண்டு கட்டுந்
தூயமுக மஞ்சளிட நிறம தாகும்
வெருகவே வீக்கமாய்க் களைப்புண்டாகும்
மிகக்காது மந்தந்தலை கனப்புண்டாகும்.”

-யூகிமுனி

- ❖ வாய் நீர் ஊறல்,
- ❖ வாய்குமட்டல்,
- ❖ நகைத்தல்,
- ❖ உணவில் வெறுப்பு,
- ❖ உண்ணிலும் செரியாமை,

- ❖ உடல் வறட்சி,
- ❖ தோல் சுருங்கி தவளை தோலை ஒத்தல் என்னும் குறிகுணங்களை காட்டி,
- ❖ கண், நகக்கண், முகம், உடலின் தோல் முதலியவகளும், சிறுநீரும் மஞ்சளிக்கும்.
- ❖ அன்றியும் இதில் உள்ளங்கால், கை, முகம், கண், உடம்பு இவை வெளுத்தல்,
- ❖ கையும் காலும் சோர்தல்,
- ❖ உடல் நடுக்கல்,
- ❖ அடிக்கடி இளைப்பு தோன்றல்,
- ❖ எரு கட்டுபட்டு தீய்ந்து வெளியாதல்,
- ❖ மிகத்தூக்கம்,
- ❖ தலைகனத்தல், முதலிய குறிகுணங்களைக் காட்டி உடல் முழுதும் மஞ்சளிக்கும்

நோய் எண்: 13

1. ஊது காமாலை
2. வறள் காமாலை
3. வளி காமாலை
4. அழல் காமாலை
5. ஐய காமாலை
6. வளிஐய காமாலை
7. அழல்ஐய காமாலை
8. முக்குற்ற காமாலை
9. மஞ்சள் காமாலை
10. அழகு காமாலை
11. செங்கமல காமாலை
12. கும்ப காமாலை
13. குன்ம காமாலை

குற்றவேறுபாடுகள்:

தீ குற்றத்தைப் பெருக்கக்கூடிய உணவுப்பொருள்களாலும், வெய்யிலில் திரிதல், இராக்கண்விழித்தல், முதலியவைகளாலும் அழல் குற்றம் மிகுந்து, அக்குற்றம் தனக்கு துணையாக ஐயத்தைக் கூட்டிக்கொண்டு, வியானனின் தொழிலை கெடுத்து இரத்தத்தின் வன்மையை கேடடையச் செய்வதால் இந்நோய் பிறக்கும். பித்துநீரும் பெருத்து இயற்கையாக கழியாது குருதியோடு கூடி, கெடுதிகளை உண்டாக்கும்.

நாடிநடை:

1. பண்பான **பித்தத்தில் சேத்து மநாடி**

   .....

   கண்காது நயனமலம் நீரு மஞ்சள்.

   (சதக நாடி)

2. சாறுமடா **பித்தமந்த வாதத்தி லேறில்**

   தளஞ்செய்யும் பாண்டு காமாலை தானும்

   (நாடி நூல்)

பித்தஜயக் கலப்பாலும், பித்தவாதக் கலப்பாலும் மஞ்சள்நோய் உற்பத்தியாகும் என்பதாம்.

மருத்துவம்:

கழிச்சலுக்கு:

1. திராட்சாதி குடிநீர்
2. கீழாநெல்லி சாற்றில், பேதியுப்பு சேர்த்து வழங்க வேண்டும்
3. சஞ்சீவி மாத்திரையை இலைக்கள்ளி சாற்றில் வழங்க வேண்டும்

உள் மருந்து:

1. **கற்கம்:**

   சீந்தில்
   கீழாநெல்லி
   கடுக்காய்
   வழங்கவும்
   கரிசாலை
   நெருஞ்சில்

   இவற்றினை அரைத்த கற்கம் 5கி, பாலுடன்

2. இளநீர் ஒன்றை கண்திறந்து அதில் 4 தேற்றான் விதையை தட்டிப் போட்டு இரவில் வைத்திருந்து காலையில் மேற்படி இளநீர் விட்டு தேற்றான் விதையை அரைத்து இளநீரில் கலக்கி அருந்தவும். இவ்வாறு 3 – 5 நாட்கள் காலை மட்டும் தரலாம்.

3. **குடிநீர் :**

கீழ்க்காய் நெல்லியின் சாறு ஓர் ஆழாக்கில் எட்டு முதல் 10 வராகனெடை பேதியுப்புக் கூட்டி சிறுதீயில் எரித்து உப்பு கரைந்த உடனே வடித்துக் காலையில் கொடுக்கக் கழியச் செய்யும்.

4. **மாத்திரை:** சாந்த சந்திரோய மாத்திரை

5. **பற்பங்கள்:**

நண்டுக்கள் பற்பம்
குக்கில் பற்பம்
சிலாசத்து பற்பம்
பலகறை பற்பம்

6. **செந்தூரங்கள்:**

காளமேக நராயண செந்தூரம்
லோகமண்டூர செந்தூரம்
வெடிஅன்னபேதி செந்தூரம்
அயசெந்தூரம்

7. **சுண்ணம்:**

நவாச்சார சுண்ணம்
வெடியுப்பு சுண்ணம்

நோய் தடுப்பு:

1. மோர், மாதுளை, கரும்புசாறு உண்ணலாம்.
2. உப்பு, புளி, நெய் எண்ணெய், முதலிய கொழுப்பு பொருள் நீக்கவும்.
3. ஆமணக்கு கொழுந்தை சாறு பிழிந்து வெள்ளாட்டுப் பாலில் கலந்து 200 மிலி வழங்கவும்

3.4. MODERN ASPECTS OF THE DISEASE

Jaundice:

Definition: Jaundice (icterus) is the yellow appearance of the skin, sclerae and mucous membranes resulting from an increased bilirubin concentration in the body fluids.

It is detected clinically when the plasma bilirubin exceeds 50 µmol/l (3mg/dl)

Hepatic Jaundice:

Hepatic causes include acute hepatitis, hepatotoxicity and alcoholic liver disease, whereby cell necrosis reduces the liver's ability to metabolise and excrete bilirubin leading to a build up in the blood.

Less common causes include primary biliary cirrhosis, Gilbert's syndrome (a genetic disorder of bilirubin metabolism which can result in mild jaundice, which is found in about 5% of the population) and metastatic carcinoma.

Jaundice seen in the newborn, known as neonatal jaundice, is common, occurring in almost every newborn as hepatic machinery for the conjugation and excretion of bilirubin does not fully mature until approximately two weeks of age.

Normal Bilirubin Metabolism:

It can be described under 4 main headings.

1.Source of Bilirubin: About 80-85% of the bilirubin is derived from the catobolism of haemoglobin preasent in senescent red blood cells.The remaining 15-20% of the bilirubin comes partly from non haemoglobin heam containing pigments such as myoglobin,catalases and cytocromes,and partly from inaffective erythropoiesis.

2. Transport of bilirubin: Bilirubin on release from macroptages circulates as unconjucated bilirubin in plasma tightly bound to albumin.

3. Hepatic phase: On coming in content with the hepatocyte surface unconjugated bilirubin is preferably metabolised in 3 steps.

I).Hepatic uptake:

Albumin bound unconjugated bilirubin upon enjoy into the hepatocytes, is disassosiated into bilirubin and albumin gets bound to cytoplasmic protein glutathione-8- transferose.

II).Conjucation and unconjucation:

Bilirubin in the blood is normally almost all unconjucated and because it is not water soluble,it is bound albumin and does not the urine.unconjucated bilirubin is conjucated by glucuronyl transferase into water soluble conjucates which are exported into the bile.

III).Secretion into bile:

Conjucated bilirubin is rapidly transported directly into bile canaliculi by energy dependent process and then excreted into the bile.

4. Intestinal phase:

Appearance of conjucated bilirubin in the intestinal lumen is followed by either direct excretors in the stool as stercobilinogen or may be metobolised to urobilinogen.

Pathophysiology:

In most cases, hyperbilirubinomia itself has little pathophysiology effect.there are two important differences between the forms of bilirubin.unconjucated bilirubin is virtually insoluble in water at physiologic pH and is tightly compleed to serum albumin.this form cannot be excreted in the urine even when blood are high.normally,a very small amount of unconjucated bilirubin is present of unbound bilirubin may diffuse into tissue, particularly the brain in infants, and produce toxic injury.

The unbound plasma fraction may increase in severe hemolytic disease or when protein binding drugs displace bilirubin from albumin.hence,hemolytic diseaseof the newborn may be lead to accumulation of unconjucated bilirubin in the brain,which can cause severe neurologic damage,referred to as kernicterous in

contrast,conjucated bilirubin is water soluble,nontoxic, and only loosely bound to albumin.because of its solubility and weak association with albumin,excess conjucated bilirubin in plasma can be excreted in urine.

Jaundice occurs when the equilibrium between bilirubin productin and clearance is disturbed by one (or) more of the foolowing mechanism:

1. Excessive production of Bilirubin.
2. Reduced Hepatocyte uptake
3. Impaired conjucation.
4. Decreased Hepatocellular excretion.
5. Impaired bile flow (Both intra hepatic and Extra hepatic)

Hepatic events

The unconjugated bilirubin then travels to the liver through the bloodstream. Because this bilirubin is not soluble, however, it is transported through the blood bound to serum albumin. Once it arrives at the liver, it is conjugated with glucuronic acid (to form bilirubin diglucuronide, or just "conjugated bilirubin") to become more water soluble. The reaction is catalyzed by the enzyme UDP-glucuronyl transferase.

This conjugated bilirubin is excreted from the liver into the biliary and cystic ducts as part of bile. Intestinal bacteria convert the bilirubin into urobilinogen. From here the urobilinogen can take two pathways. It can either be further converted into stercobilinogen, which is then oxidized to stercobilin and passed out in the feces, or it can be reabsorbed by the intestinal cells, transported in the blood to the kidneys, and passed out in the urine as the oxidised product urobilin. Stercobilin and urobilin are the products responsible for the coloration of feces and urine, respectively.

Signs and symptoms mentioned in various sources for Hepatocellular jaundice:

- Liver diseases
- Jaundice
- Increased plasma bilirubin levels
- Anorexia

- Nausea
- Vomiting
- Fatigue
- Insomnia
- Yellow colored sclera
- Yellow colored urine
- Pain in abdomen
- Fever
- Diarrhoea
- Hematemesis

Hepatocellular diseases

Viral hepatitis

- Hepatitis A, B, C, D, and E
- Epstein-Barr virus (EBV)
- Cytomegalovirus (CMV)
- Herpes simplex

Drug toxicity

- Classified as either predictable or unpredictable
- Predictable reactions are dose-dependent and affect all patients who ingest toxic dose; classic example is acetaminophen hepatotoxicity.
- Unpredictable or idiosyncratic drug reactions are not dose-dependent and occur in few patients.
- Many drugs can cause idiosyncratic hepatic injury.

Environmental toxins include:

- Industrial chemicals such as vinyl chloride
- Herbal preparations containing pyrrolizidine alkaloids (e.g., Jamaica bush tea) and kava kava
- Mushrooms *Amanita phalloides* or *Amanita verna* that contain highly hepatotoxic amatoxins

Alcoholic hepatitis

Can be differentiated from viral and toxin-related hepatitis by pattern of aminotransferase in serum.

Viral Hepatitis:

Viral hepatitis is almost always caused by one or other of the specific hepatitis viruses; hepatitis due to other viruses accounts for only about 1-2% of cases. All these viruses give rise to illnesses which are similar in their clinical and pathological features and which are frequently anicteric or asymptomatic.

Alcoholic (Ethanolic) liver disease:

In many societies alcohol is the most common cause of chronic liver disease. Alcohol is metabolised almost exclusively in the liver. It is first converted to acetaldehyde, mainly by mitochondrial enzyme alcohol dehydrogenase but also by the mixed-function oxidase enzymes of the smooth endoplasmic reticulum. Alcohol is a powerful inducer of the mixed-function oxidases.

The hepatic lesions of alcoholic liver disease are attributable directly to alcohol. The risk of developing alcoholic liver disease is related to alcohol intake above 30 g(3units) in men and 20 g(2units)in women. More than 5 years of drinking, and usually more than 10 years, are required to produce alcoholic chirrhosis.

Fatty change is attributed to an impaired excretion and enhanced synthesis of triacylglycerol by hepatocytes. The development of alcoholic hepatitis, fibrosis and cirrhosis is much more obsure. Biochemical mechanisms involving the production of toxic metabolites, called adducts, during the conversion of acetaldehyde to acetate and an immune reaction to liver cells altered by alcohol may be involved in these forms of liver damage. Alcohol causes several different lesions in the liver which can occur together in any combination.

FATTY LIVER

Accumulation of fat in a liver beyond the level which is normally encountered may be a result of a normal physiological response to increased peripheral lipolysis, obesity or the action of hepatotoxins.

Fatty liver disease

Fatty liver disease can range from fatty liver alone (steatosis) to fatty liver associated with inflammation (steatohepatitis). This condition can occur with the use of alcohol (alcohol-related fatty liver) or in the absence of alcohol (non-alcoholic fatty liver disease [NAFLD].

Fatty liver can be associated with the use of alcohol. This may occur with as little as 10 oz of alcohol ingested per week. Identical lesions also can be caused by other diseases or toxins. Fatty change in the liver results from excessive accumulation of lipids within hepatocytes. Simple fatty liver is believed to be benign, but NASH can progress to cirrhosis and can be associated with hepatocellular carcinoma. The main risk factors for simple fatty liver (NAFLD) and NASH are obesity, diabetes, high triglyceride levels, or a high fat diet.

Non-alcoholic fatty liver disease:

NAFLD is one cause of a fatty liver, occurring when fat is deposited (steatosis) in the liver not due to excessive alcohol use. It is related to insulin resistance and the metabolic syndrome and may respond to treatments originally developed for other insulin-resistant states (e.g. diabetes mellitus type 2) such as weight loss, metformin and thiazolidinediones. Non-alcoholic steatohepatitis (**NASH**) is the most extreme form of NAFLD this being regarded as a major cause of cirrhosis of the liver of unknown cause.

Exams and Tests Physical examination:

- Nutritional assessment
- Yellowing of the sclerae is usually the first detectable sign of jaundice.

- Darkening of urine
- Skin examination for icterus
- Stigmata of chronic liver disease
- Abdominal examination
- Enlarged and tender liver
- Fluid in the abdomen (ascites) that can become infected

Blood tests

These may initially include

- Complete blood count – TC ,DC, ESR, Cholesterol
- Liver function test
- In women, a pregnancy test may be obtained.

Urine alysis: Urine analysis for bile salts and bile pigments

Laboratory tests:

- Abdominal ultrasound
- Autoimmune blood markers
- Hepatitis virus serologies
- Liver function tests
- Liver biopsy to check for liver damage
- Paracentesis if fluid is in abdomen

Tests for Liver Function

Bilirubin:

Bilirubin is one of the most important factors indicative of hepatitis. It is a red-yellow pigment that is normally metabolized in the liver and then excreted in the urine. In patients with hepatitis, the liver cannot process bilirubin, and blood levels of this substance rise. High levels of bilirubin cause the yellowish skin tone known as jaundice.

Liver Enzymes (Aminotransferases):

Enzymes known as aminotransferases, including aspartate (AST) and alanine (ALT), are released when the liver is damaged. Measurements of these enzymes, particularly ALT, are the least expensive and most noninvasive tests for determining severity of the underlying liver disease and monitoring treatment effectiveness. Enzyme levels vary, however, and are not always an accurate indicator of disease activity.

Alkaline Phosphatase (ALP): High ALP levels can indicate bile duct blockage.

GGT (gamma glutamyl transpeptidase) is often elevated in those who use alcohol or other liver-toxic substances to excess.

Serum Albumin:

Serum albumin measures protein in the blood (low levels indicate poor liver function). **Total protein.** Serum total protein measures protein in the blood (low levels indicate poor liver function).

Prothrombin Time (PT):

The PT test measures in seconds the time it takes for blood clots to form (the longer it takes the greater the risk for bleeding

3.5. EARLIER STUDIES ON HEPATOPROTECTIVE

Hydro alcoholic extract of tubers of Momordica tuberosa was subjected to preliminary phytochemical screening and evaluated for in vitro and in vivo antioxidant and hepatoprotective activity against CCl_4 induced liver damage in rats. Pretreatment with 70% ethanolic extract of M. tuberosa reversed CCl_4 induced elevation of levels of serum biomarkers to near normal levels, suggesting that the tubers of M. tuberosa possess hepatoprotective property and this property may be attributed to the antioxidant property of the plant. (Pramod Kumar el al, 2008).

Hepatoprotective and antioxidant effects of tender coconut water (TCW) were investigated in carbon tetrachloride (CCl_4)-intoxicated female rats. Liver damage was evidenced by the increased levels of serum glutamate oxaloacetate transaminase

(SGOT), serum glutamate pyruvate transaminase (SGPT) and decreased levels of serum proteins and by histopathological studies in CCl_4 intoxicated rats. Increased lipid peroxidation was evidenced by elevated levels of thiobarbituric acid reactive substance (TBARS) viz, malondialdehyde (MDA), hydroperoxides (HP) and conjugated dienes (CD), and also by significant decrease in antioxidant enzymes activities, such as superoxide dismutase (SOD), catalase (CAT) and also reduced glutathione (GSH) content in liver. On the other hand, CCl_4 intoxicated rats treated with TCW retained almost normal levels of these constituents. Decreased activities of antioxidant enzymes in CCl_4 intoxicated rats and their reversal of antioxidant enzyme activities in TCW treated rats, shows the effectiveness of TCW in combating CCl_4 induced oxidative stress. Hepatoprotective effect of TCW is also evidenced from the histopathological studies of liver, which did not show any fatty infiltration or necrosis, as observed in CCl_4 intoxicated rats. (Anthony Loperito Loki and Rajamohan, 2003)

4. MATERIALS AND METHODS

4.1. Preparation of *Chara parpam*:

1. ***Navacharam.(*Ammonium chloride)**
2. ***Vediyuppu* (Potassium nitrate)**
3. ***Aadathodi* (*Justicia adatoda)***

4.1.1. Navacharam: Raw Navacharam was bought from the TAMCOL sales counter, Arumbakkam, Chennai -106. The raw material was identified and authenticated by PG Gunapadam dept.Govt.Siddha Medical college, Chennai. 500gm Navacharam was taken and purification process was done. After purification weight of the drug was 450gm.

4.1.2. Vediyuppu: Raw Vediyuppu was bought from the TAMCOL sales counter, Arumbakkam, Chennai -106. The raw material was identified and Authenticated by PG Gunapadam dept.Govt.Siddha Medical college, Chennai. 500gm Vediyuppu was taken and purification process was done. After purification weight of the drug was 450gm.

Purification of *Navacharam:* 100gm of Navacharam is dissolved in cow's urine then filtered and boiled and dried in sunlight.

Purification of *Vediyuppu:*With 100gm of vediyuppu add 500 ml of water and heat it, when boiled add one egg white to it,dust particles will accumulated in the upper surface should be removed and filtered. Next day it is dried in sunlight. This above said procedure is repeated for 7 times.

4.1.3. Aadathodi Juice: The juice was collected from the leaves of the plants around 6a.m at Vadalur, Cuddalore(dt). Tamilnadu.

Method of Preparation: Fine powders of Navacharam and Vediyuppu. These drugs are mined together and grounded with Aadathodai juice for 12 hours and made in small cakes (villai) and dried in sunlight. These cakes are then kept in a suitable pan of mud vessel and sealed with 7 layers of clay cloth (seelai Mann). Then the sealed vessel (kavasam) is dried and burned for 12 hours. Allow it to cool at room temperatures then the kavasam is opened. The material found in white in colour which is called as Chara Parpam.

PREPARATION OF *CHARA PARPAM*.

Fig.8. Purified Charam

Figure.9. Purified Vediyuppu

Figure.10. Aadathoda Juice

Figure.11. Chara Parpam

Figure.12. Charaparpam

4.2. STANDARDIZATION OF THE DRUG

4.2.1. Physico-chemical analysis

4.2.1.1. Ash and acid insoluble ash:

To the ash add 1:5 Hcl: Distilled water 15 ml boil, cooled and then filtered using whatman filter paper (No.41) repeat 3 to 4 times till the yellow colour disappear or colourless, then remove the filter paper and add to the filter to the original dish and keep it in the muffle furnace at 600^{o} C and take constant weight and calculate the acid insoluble ash value.

$$\text{Acid insoluble ash (\%)} = \frac{\text{Weight of acid insoluble residue x 100}}{\text{Weight of the sample}}$$

Acid insoluble residue = Acid insoluble ash value – Empty weight of the dish

Loss on drying:

3gm of the drug is heated in a hot oven at 105^{0} c to constant weight. The % of weight was calculated.

Loss on drying value at 105^{0} c - 10.96 %w/w

Potential of Hydrogen (ph):

The pH scale is logarithmic and runs from 0.0 to 14.0 with 7.0 being neutral. Readings less than 7.0 indicate acidic solutions, while higher readings indicate alkaline or base solutions.

4.2.2 SCANNING ELECTRON MICROSCOPE:

A **scanning electron microscope** (**SEM**) is a type of electron microscope that produces images of a sample by scanning it with a focused beam of electrons. The electrons interact with electrons in the sample, producing various signals that can be detected and that contain information about the sample's surface topography and composition. The electron beam is generally scanned in a raster scan pattern, and the beam's position is combined with the detected signal to produce an image. SEM can achieve resolution better than 1 nano meter. Specimens can be observed in high vacuum, low vacuum and in environmental SEM specimens can be observed in wet condition.

Figure.13

Resolution **:** 1.2 nm gold particle separation on a carbon substrate

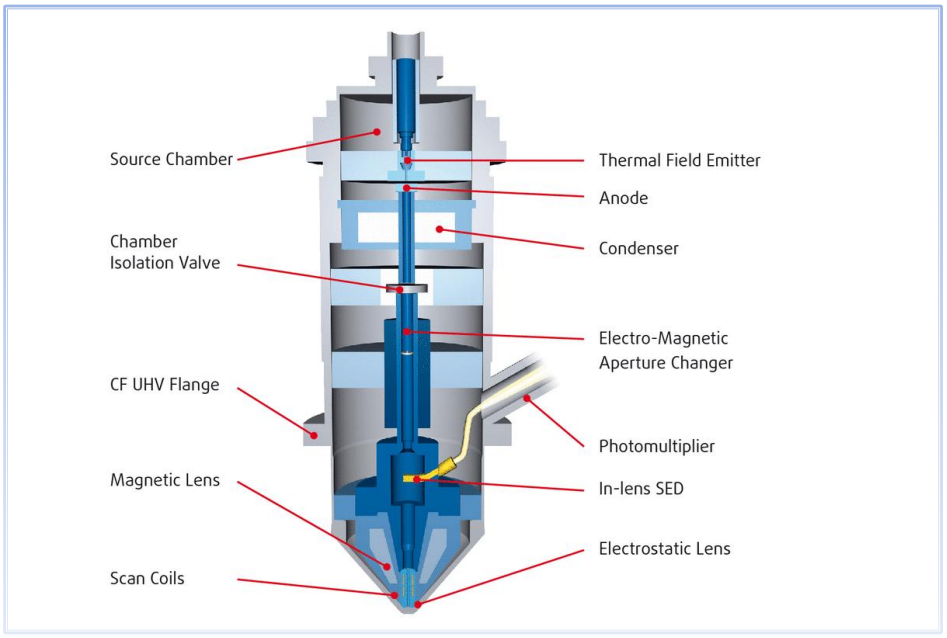
Magnification: From a min of 12 x to greater than 1, 00,000 X

The SEM has a large depth of field, which allows a large amount of the sample to be in focus at one time. The SEM also produces images of high resolution, which means that closely spaced features can be examined at a high magnification. Preparation of the samples is relatively easy since most SEM one require the sample to be conductive.

The combination of higher magnification, larger depth of focus, greater resolution, and easy of sample observation marks the SEM one of the most heavily used instruments in research areas today.

MECHANISMS:

Figure.14. Mechanism of SEM



4.2.3. FOURIER TRANSFORM INFRARED SPECTROSCOPY (FTIR):

Insrumental details:

Figure.15

Model	**:**	**Spectrum one: FT-IR Spectrometer**
Scan Range	**:**	**MIR 450-4000 cm-1**
Resolution	**:**	**1.0 cm-1**
Sample required	**:**	**50 mg, solid or liquid.**

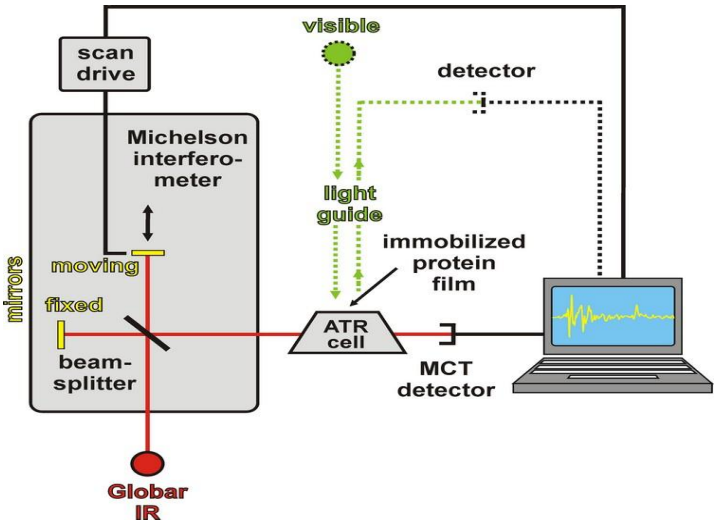
Fourier Transform Infrared Spectroscopy (FTIR) is an analytical technique used to identify mainly organic materials. FTIR analysis results in absorption spectra which provide information about the chemical bonds and molecular structure of a material. The FTIR spectrum is equivalent to the "fingerprint" of the material and can be compared with cataloged FTIR spectra to identify the material.

Infrared spectrum is useful in identifying the functional groups like -OH, -CN, -CO, -CH, -NH2, etc. Also quantitative estimation is possible in certain cases for chemicals, pharmaceuticals, petroleum products, etc. Resins from industries, water and rubber samples can be analyzed.

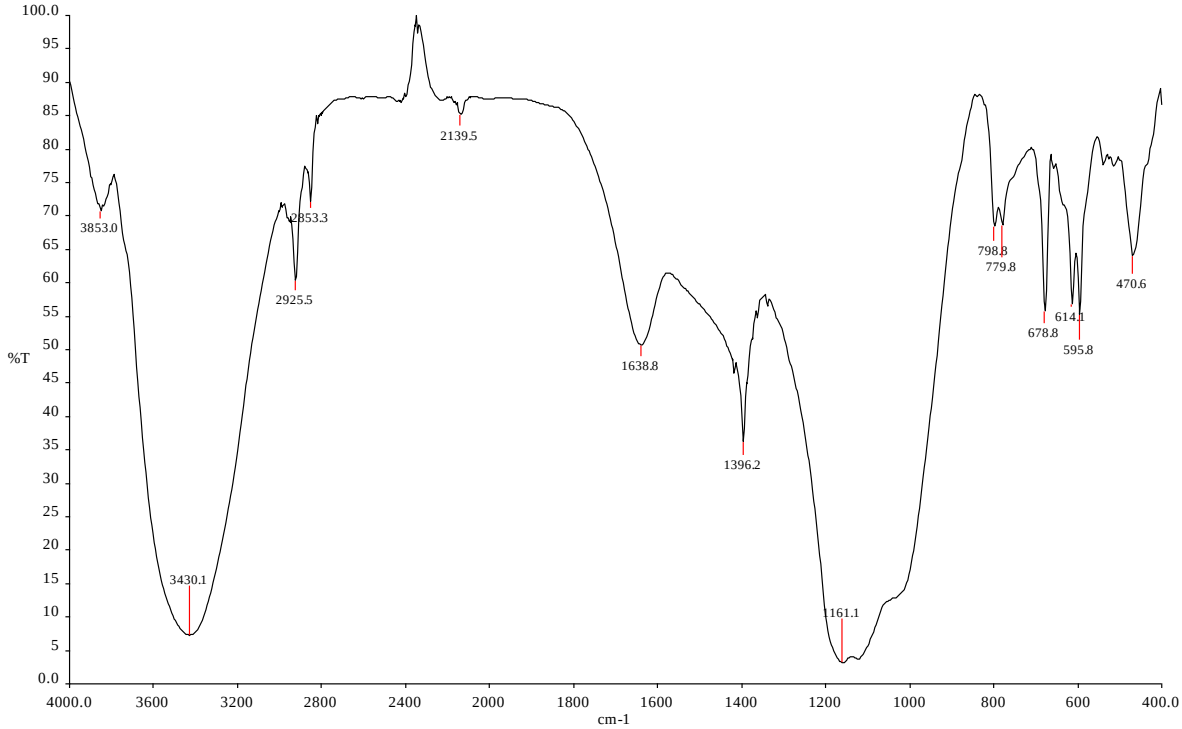
The drug sample was analyzed by the FTIR to identify the chemical bonds and molecular structure of a material.

Mechanisam of FTIR:

Figure.16.



FTIR Study on *Chara Parpam:*



4.2.4. Bio -chemical analysis:

Preparation of extract of test drug:

Add 5 gm of ***Chara parpam*** to 50ml of distilled water. The solution is boiled for 20 minutes, then it is cooled and then filtered in a 100ml volumetric flask. Use the Extract for the following tests.

S.No	Experiment	Observation	Inference
1.	**Test for Reducing Sugar :** To 5ml of Benedicts qualitative reagent, add 10 drops of extract,	Absence of Green Precipitate	Absence of Reducing Sugar
2.	**Test for Starch :** To 5 ml of extract add 2ml of acetic acid and then add few drops	Absence of Blue Colour	Absence of Starch
3.	**Test for Proteins :** To 2 ml of extract, add 2ml of 5% Sodium Hydroxide mix and add 2	Absence of Violet Colour	Absence of Proteins
4.	**Test for amino Acid :** Place 2 drops of extract on a filter paper and allow to dry well. Then	Absence of Violet Colour	Absence of Amino Acid
5.	**Test for Albumin :** To 2 ml of extract, add 2ml of Esboch's reagent.	Absence of Yellow Precipitate	Absence of Albumin
6.	**Test for Phosphate :** To 2ml of extract, add 2ml of ammonium Molybdate solution	Absence of Yellow Precipitate	Absence of Phosphate
7.	**Test for Sulphate** : To 2 ml of extract add 2ml of 4% ammonium oxalate solution.	Presence of White Precipitate	Presence of Sulphate
8.	**Test for Chloride :** Add 2ml of extract to dilute nitric acid till the effervescence ceases.	Presence of Cloudy White Precipitate	Presence of Chloride

9.	**Test for Iron :** To 2ml of extract, add 2ml of ammonium thiocynate solution	Presence of Red Colour	Presence of Iron
10.	**Test for Calcium :** To 2 ml of extract, add 2 ml of 4% ammonium Oxalate Solution.	Presence of White Precipitate	Presence of Calcium
11.	**Test for Sodium :** Make a paste with 2 pinches of the sample with Hcl and Introduce	Absence Yellow Flame	Absence of Sodium
12.	**Test for Potassium :** Add a pinch of the sample to 2 ml of Sodium Nitrate Solution. Then	Presence of Yellow Precipitate	Presence of Potassium

4.3. TOXICOLOGICAL STUDY ON CHARA PARPAM

Animals:

Mice of either sex weighing 25-30g and rats weighing 210-240g were obtained from the animal house of Vels University. The animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28^{0}C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum. Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group. The animals were acclimatized for one week under laboratory conditions.

Acute Toxicity Study:

Acute oral toxicity test for the Chara Parpam was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been

administered, food was withheld for a further 2 hours in mice. The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal.

Observation of toxicity signs: General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

Sub Acute Toxicity:

In a 28-days sub acute toxicity study, twenty four either sex rats were divided into four groups of 6 rats each. Group I that served as normal control was administered with distilled water (p.o.) while groups II, III and IV were administered daily with the Chara Parpam (p.o.) for 28 days at a dose of 2.5, 5.0 and 10.0mg/kg respectively. The animals were then observed daily for gross behavioural changes and any other signs of subacute toxicity. The weight of each rat was recorded on day 0 and weekly throughout the course of the study, food and water consumption per rat was calculated. At the end of the 28 days they were fasted overnight, each animal was anaesthetized with diethylether, following which they were then dissected and blood samples were obtained by cardiac puncture into heparinised tubes. The blood sample collected from each rat was centrifuged with 3000 X g at 4^{o}C for 10 min to separate the serum and used for the biochemical assays.

Hematological and blood biochemical analyses:

At the end of the study, all animals were kept fasted for 16-18 h and then anesthetized with anesthetic ether on the 28th day. Blood samples for hematological and blood chemical analyses were taken from retro orbital vein. Heparinized blood samples were taken for determining complete blood count (white blood cell count, differential white blood cell count, platelet count, red blood cell count, hematocrit,

and hemoglobin) by semiautomated hematology analyzer. The serum from non-heparinized blood was carefully collected for blood chemistry and enzyme analysis glucose, creatinine, total protein, albumin, total and direct bilirubins, serum glutamate-oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP)) were automatically determined using autoanalyzer.

Necropsy:

All rats were sacrificed after the blood collection. The positions, shapes, sizes and colors of internal organs were evaluated. The Spleen, Testes, Pancrea, Lung, Liver, Brain, Heart, Stomach, Intestine, Bone, Ovary, and Kidney tissues were excised from all rats to visually detect gross lesions, and weighed to determine relative organs' weights and preserved in 10% neutral formalin for histopathological assessment. The tissues were embedded in paraffin, and then sectioned, stained with haematoxylin and eosin and were examined microscopically.

Statistical analysis

Values were represented as mean ± SEM. Data were analysed using one-way analysis of variance (ANOVA) and group means were compared using the Tukey-Kramer Multiple Comparisms Test using GraphPad Instat-V3 software. P values < 0.05 were considered significant.

4.4. PHARMACOLOGY STUDY.

Hepatoprotective activity of chara parpam in ccl_4 induced rats:

Materials and methods

Chemicals

Estimation Kits for AST, ALT, ALP, T.P, etc were obtained from SPAN diagnostics and Standard drug Silymarin and hepatotoxin CCL_4 (Sigma-aldrich chemical Pvt. Ltd., Bangalore.) were used in the present study.

Stock Solution

Animals

Wistar albino rats (150-200 g) and Mice of either sex weighing 25-30g were procured from animal housing facility of School of Pharmaceutical Sciences, Vels University, Chennai and used for the study. The animals were housed in well ventilated cage and animals had 12 hours day and night schedule with temperature between 28 ±20C. The animals were housed in large spacious hygienic cages during the course of the experimental period. The animals were allowed free access to standard laboratory pellets and drinking water ad libitum. The study has got the approval from the Institutional Animal Ethical Committee (IAEC) of Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA). (Approval number: XIII/VELS/PCOL/01/2000/CPCSEA/IAEC/08.08.2012).

Determination of acute oral toxicity

Acute toxicity of Chara Parpam was done according to the OECD guidelines No.425. The overnight fasted mice were given in various doses (2000, 1000, 500, 250, 100 and 50mg/kg b.w.), the animal were observed continuously for the first two hours and at 24 hours to detect changes in behavioural responses and also for tremors, convulsion, salivation, diarrhoea, lethargy, sleep, coma and also were monitored up to 14 days for the toxic symptoms and mortality.

Evaluation of Hepatoprotective activity

CCL_4 induced hepatotoxicity in rats model was used for evaluation of hepato-protective activity for the Chara Parpam. Animals were divided into five groups, each group containing six animals.

Group I (normal) received distilled water or 2% CMC for 14 days. Group II (Control) received CCl_4 1ml/kg, i. p. 1:1 dilution with coconut oil on 5th day. Groups III-IV, received Chara Parpam (5mg/kg and 10mg/kg p.o) for 14 days and CCl_4 induction on 5th day. Group V received standard marketed drug silymarin (25mg/kg per day, p.o.) for 14 days and CCl_4 induction on 5^{th} day.

After 14 days of experimental period blood sample had been collected individually for all the animals by retro-orbital puncture method and the blood was allowed to clot for 30 min; serum was separated by centrifuging and was used for various parameter estimations. Later all the animals were sacrificed by cervical dislocation, liver samples were collected and the individual weights of the livers were estimated. For histopathological study, liver tissue was quickly removed after autopsy and fixed in 10% formalin in saline.

Biochemical parameters studied

The activities of serum glutamate pyruvate transaminase, and serum glutamate oxaloacetate transaminase were estimated using standard methods. Estimation of serum ALP, serum bilirubin and electrolytes were also carried out to assess the acute hepatic damage caused by CCl_4.

Statistical analysis

The data obtained from the study were subjected to statistical analysis by one way ANOVA followed by Dunnet ‘t’ test, and results were expressed in terms of Mean±SEM values. Statistical analysis was performed using INSTAT- V3 Software programme.

4.5. CLINICAL ASSESSMENT:

Liver disease is at a standstill a international health problem. Regrettably conventional or synthetic drugs used in the treatment of liver diseases are inadequate and sometimes can have serious side effects. (Manokaran et al, 2008) In view of severe unwanted side effects of synthetic agents, there is growing focus to follow systematic research methodology and to evaluate scientific basis for the herbo mineral drug that are claimed to possess hepatoprotective activity.The pre clinical study of this drug has been showed the marked hepatoprotective efficacy. The clinical study was conducted to establish the efficacy and safety of the *Chara Parpam* for *Kamalai.*

Objectives:

To evaluate the Hepatoprotective effect of *Chara Parpam.*

- To discover the efficacy and safety of *Chara Parpam* in patients with liver disorder.

Design of the study:

Open clinical trial Phase II B

Study centre:

Arignar Anna Government Hospital of Indian Medicine and Homeopathy, Arumbakkam, Chennai – 106

Study participants:

Both men and women and members of all races and ethnic groups are eligible for this trial. Treatment was administered on an inpatient/outpatient basis. The patients were selected from the In-patient and Out-patient department of Arignar Anna Government Hospital of Indian Medicine and Homeopathy, Chennai – 106.

Number of subjects:

Number of participants will be 50.

Selection:

. 50 patients from both sexes of various age groups were selected for clinical trial. Among 40 patients were treated as out-patients, 10 patients were treated as in – patients. The selection was based on the including and excluding criteria. They were clinically diagnosed on the basis of siddha principles with modern laboratory findings

Registration process:

To register a patient, the following documents have been produced.

- Copy of required laboratory tests
- Signed patient consent form
- Other appropriate forms (e.g., Eligibility Screening Worksheet, Registration Form).

Then I verified the eligibility and assigned a patient study number, drug dose and registered the patient on the study.

Criteria for inclusion:

Patients with liver disease are eligible for entry to the trial if the following criteria are satisfied.

- Co operative patients
- The previous drug regimen if any have been with held for 24 hours before the clinical trial.

Criteria for exclusion:

- AIDS
- Malignancy
- Pregnant and lactating women
- TB
- Renal diseases
- Cardio vascular disorder
- Age below 10 year
- Syphilis
- The patient requires systemic steroid for the control of symptoms

Withdrawal criteria:

Patients were removed from study when any of the criteria listed below applies. In the absence of treatment delays due to adverse events, treatment may continue for 2 cycles or until one of the following criteria applies:

- Disease progression,
- Deterioration of vital signs with cardiac, respiratory, hepatic, renal and CNS changes.
- Intercurrent illness that prevents further administration of treatment,
- Unacceptable adverse event(s),
- Patient decides to withdraw from the study, or
- General or specific changes in the patient's condition render the patient unacceptable for further treatment in the judgment of the investigator.

Routine examination and assessment:

The full details of history and physical examination of the patients were recorded as per the proforma .The clinical assessment was done initially at the end of 7 days, 14 days and 21 days during treatment and at the end of the 21 days follow up was done. The laboratory investigation and the physiological parameters werew recorded initially at the end of the treatment and at the end of follow up as per the proforma.

Dosage:

The trial drug *Chara Parpam* was given in the dose of 130 mg to 260 mg with Seeraga kudineer depending upon the severity of the case.

Administration of the drug:

Form of the medicine	: *Parpam*
Route of Administration	: Enteral
Dose	: 130 – 260 mg
Anubanam (Vehicle)	: Seeraga kudineer
Times of Administration	: Two times a day; after food
Duration	: 7 weeks

Diet restriction and Medical advice:

The patients were instructed to follow fat free, salt free easily digestible foods.

- They were advised to take tender coconut, sugar cane juice and vegetables like radish, juice of plantain stem.
- The patient was advised to cold damp climate.
- The patient was advised to take rest. But prolonged immobilization should be avoided.
- The clinical improvement was observed and recorded daily in the proforma of case sheet.

Trail conduct:

The study was conducted in compliance with the protocol approved by the Institutional Review Board, and according to Good Clinical Practice standards. No deviation from the protocol was implemented without the prior review and approval of the IRB except.

Classification of results:

1. **Good Response**
    a. Relief of Symptoms above 70%
    b. Laboratory parameter findings towards normalcy.
2. **Fair Response**
    a. 50% to 70% relief in symptoms.
    b. Significant improvement in laboratory parameter.

3. **Poor Response**

   20% to 49% relief in symptoms and minimal improvement in laboratory parameters.

4. **No Response**

   No relief in symptoms and no significant improvement in laboratory parameters.

Follow up:

Assessment will take for every three days before treatment and after treatment. During this period clinical assessment and laboratory investigation was carried out.

Statistical analysis:

The data will be tabulated and analyzed by students 'T' test.

Ethical review:

The protocol and any amendments were submitted to Govt siddha medical college, Chennai – 106 (IEC) and got formal approval to conduct the study. The decision of the IEC concerning the conduct of the study was made in writing to the investigator. All subjects for this study was provided a consent form describing this study and provided sufficient information for subjects to make an informed decision about their participation in this study. This consent form was submitted with the protocol for review and approval by the IEC. The formal consent of a subject, using the IEC-approved consent form, was obtained before that subject was submitted to any study procedure. This consent form was signed by the subject or legally acceptable surrogate, and the investigator-designated research professional obtaining the consent.

CLINICAL STUDY ON CHARAPARPAM IN – OUT PATIENTS DEPT IN THE MANAGEMENT OF KAMALAI.

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD										Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb gm	Sgr mg/dl	Ur mg/dl	Blood CL	Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
1.	1818	Amudhan 18/Male	Anorexia, Nausea, Vomiting, skin itching, Fatigue, Insomnia, Yellow colored sclera.	24.7.12 To 24.8.12	BT	9200	52	45	3	17	19	13	101	21	173	NIL	NIL	PCS	Satisfactory
					AT	9200	50	47	3	14	24	13	96	18	168	NIL	NIL	NIL	
2.	3436	Babu 44/Male	Anorexia, Nausea, Vomiting, skin itching, Fatigue, Insomnia, Yellow colored sclera.	1.8.12 To 31.8.12	BT	9700	57	39	4	15	24	11	94	24	158	NIL	NIL	FPC	Good
					AT	9700	59	36	5	14	20	11.3	96	22	152	NIL	NIL	NIL	
3.	2857	Kalaivani 45/Female	Anorexia, Nausea, Vomiting, skin itching, Fatigue, Insomnia, Yellow colored sclera.	2.8.12 To 30.8.12	BT	9100	58	39	4	26	44	10.5	130	23	165	NIL	NIL	FPC	Good
					AT	9200	55	40	5	27	43	10.6	117	20	154	NIL	NIL	NIL	
4.	6798	SathiyaThilaga 25/Female	Anorexia, Nausea, Vomiting, skin itching, Fatigue, Insomnia, Yellow colored sclera.	8.8.12 To 5.9.12	BT	8600	61	34	5	10	15	12	95	24	159	NIL	NIL	FPC	Good
					AT	8700	58	39	3	14	20	12	93	18	165	NIL	NIL	NIL	
5.	4673	Vasanth 37/male	Anorexia, Nausea, Vomiting, skin itching, Fatigue, Insomnia, Yellow colored sclera.	8.8.12 To 4.9.12	BT	9100	60	34	6	15	33	10	85	21	158	NIL	NIL	NIL	Moderate
					AT	9100	61	37	2	15	30	10.5	86	19	144	NIL	NIL	NIL	

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD										Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb Gm	Sgr mg/dl	Ur mg/dl	Blood CL	Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
6.	4534	Seetharam 46/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	9.8.12 To 7.9.12	BT	9300	53	42	5	6	13	10	73	21	164	NIL	NIL	PCS	Good
					AT	9300	52	44	4	6	13	10	72	19	163	NIL	NIL	NIL	
7.	7089	Mahendran 27/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	16.8.12 To 14.9.12	BT	8700	58	37	5	14	24	13.5	88	19	190	NIL	NIL	NIL	Good
					AT	8700	58	37	5	14	20	13.6	86	20	185	NIL	NIL	NIL	
8.	6897	Sudha 56/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	16.8.12 To 12.9.12	BT	9100	56	38	6	13	16	11	101	20	150	NIL	NIL	FPC	Good
					AT	9200	55	40	5	13	16	11	96	21	144	NIL	NIL	NIL	
9.	5768	Maran 22/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	18.8.12 To 16.9.12	BT	9600	58	38	4	10	15	10	90	23	159	NIL	NIL	NIL	Good
					AT	9600	58	38	4	10	15	10.5	89	20	168	NIL	NIL	NIL	
10.	8609	David 40/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	20.8.12 To 17.9.12	BT	9800	55	40	5	15	20	13	95	21	161	NIL	NIL	FPC	Good
					AT	9800	56	39	5	15	20	13	94	18	157	NIL	NIL	NIL	

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD									Blood CL	Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb gm	Sgr mg/dl	Ur mg/dl		Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
11.	8978	Ragu 28/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	22.8.12 To 19.9.12	BT	9300	57	38	5	13	25	12.5	95	26	168	NIL	NIL	PCS	Good
					AT	9200	57	38	5	13	25	12.5	96	28	162	NIL	NIL	NIL	
12.	9032	Ravi 40/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	22.8.12 To 21.9.12	BT	8700	55	41	4	20	40	10	128	21	171	NIL	NIL	NIL	Good
					AT	8800	53	41	6	20	45	10	103	19	164	NIL	NIL	NIL	
13.	9098	Madhavan 52/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	22.8.12 To 19.9.12	BT	10000	59	36	5	18	20	14	105	18	170	NIL	NIL	FPC	Good
					AT	10000	58	37	5	18	20	14	106	20	173	NIL	NIL	NIL	
14.	253	Kumar 22/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	25.8.12 To 21.9.12	BT	8500	63	30	7	25	20	13	85	23	159	NIL	NIL	NIL	Moderate
					AT	8700	63	30	7	25	20	13	88	19	165	NIL	NIL	NIL	
15.	162	Lakshmi 45/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	25.8.12 To 21.9.12	BT	9900	54	41	5	20	30	12	95	16	162	NIL	NIL	FPC	Good
					AT	9900	53	42	5	20	30	13	92	19	166	NIL	NIL	NIL	

CLINICAL STUDY ON CHARAPARPAM IN – OUT PATIENTS DEPT IN THE MANAGEMENT OF KAMALAI.

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD									Blood CL	Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb Gm	Sgr mg/dl	Ur mg/dl		Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
16.	294	Vaithilingam 49/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	25.8.12 To 21.9.12	BT	9500	57	38	5	20	43	12	85	19	160	NIL	NIL	PCS	Mild
					AT	9600	58	37	5	20	43	12.4	85	21	159	NIL	NIL	FPC	
17.	355	Visalatchi 42/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	26.8.12 To 23.9.12	BT	9100	56	38	6	14	20	12.7	95	20	158	NIL	NIL	NIL	Good
					AT	9200	55	39	6	14	20	12.7	90	22	160	NIL	NIL	NIL	
18.	405	Kumari 35/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	26.8.12 To 24.9.12	BT	9500	58	37	5	15	25	10.2	86	21	159	NIL	NIL	FPC	Poor
					AT	9700	59	38	3	15	25	10.2	85	20	160	NIL	NIL	NIL	
19.	776	Santhosh 26/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	26.8.12 To 23.9.12	BT	8100	63	34	3	20	40	12	99	21	155	NIL	NIL	NIL	Good
					AT	8200	63	34	3	20	40	12	94	19	166	NIL	NIL	NIL	
20.	2457	Mohammad mustafa 51/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	2.9.12 To 28.9.12	BT	9400	62	34	4	12	24	13.5	108	28	168	NIL	NIL	PCS	Moderate
					AT	9400	61	35	4	12	24	13.5	104	26	165	NIL	NIL	FPC	

CLINICAL STUDY ON CHARAPARPAM IN – OUT PATIENTS DEPT IN THE MANAGEMENT OF KAMALAI.

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD									Blood CL	Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb Gm	Sgr mg/dl	Ur mg/dl		Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
21.	3001	Thangaraj 29/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	5.9.12 To 3.10.12	BT	9200	56	38	6	14	30	14	99	21	162	NIL	NIL	PCS	Good
					AT	9300	56	38	6	14	30	14	98	19	158	NIL	NIL	FPC	
22.	4347	Dhandapani 40/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	8.9.12 To 5.10.12	BT	10300	55	40	5	10	24	13	120	24	153	NIL	NIL	PCS	Good
					AT	10200	56	39	5	10	24	13.2	116	20	155	NIL	NIL	NIL	
23.	5675	Ravisankar 45/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	17.9.12 To 15.10.12	BT	8600	63	34	3	11	24	12.8	85	21	162	NIL	NIL	FPC	Good
					AT	8800	63	34	3	11	25	12.8	88	18	164	NIL	NIL	NIL	
24.	5674	Vinoth 55/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	22.9.12 To 19.10.12	BT	10000	57	38	5	22	30	10	115	20	157	NIL	NIL	PCS	Moderate
					AT	10100	58	37	5	22	30	10.3	112	19	160	NIL	NIL	FPC	
25.	9555	Punitha 44/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	22.9.12 To 21.10.12	BT	9700	60	34	6	35	60	11.5	77	18	157	NIL	NIL	NIL	Good
					AT	9800	61	33	6	30	55	11.5	75	21	160	NIL	NIL	NIL	

CLINICAL STUDY ON CHARAPARPAM IN – OUT PATIENTS DEPT IN THE MANAGEMENT OF KAMALAI.

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD										Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb Gm	Sgr mg/dl	Ur mg/dl	Blood CL	Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
26.	2198	Rajapriyan 33/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	29.9.12 To 26.10.12	BT	8200	62	35	3	12	25	15	88	21	163	NIL	NIL	FPC	Good
					AT	8300	62	34	4	12	25	15	87	23	161	NIL	NIL	NIL	
27.	3651	Geetha devi 28/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	3.10.12 To 31.10.12	BT	9500	59	38	3	13	15	13	85	19	163	NIL	NIL	PCS	Good
					AT	9400	59	38	3	13	15	13.4	84	20	161	NIL	NIL	NIL	
28.	5707	Saravanan 24/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	10.10.12 To 6.11.12	BT	8900	65	31	4	14	22	15	98	20	159	NIL	NIL	NIL	Good
					AT	9000	65	31	4	14	22	15	93	18	160	NIL	NIL	NIL	
29.	3897	Deepak 31/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	4.10.12 To 2.11.12	BT	9600	59	38	3	10	15	14	98	24	167	NIL	NIL	PCS	Poor
					AT	9600	59	37	4	10	15	14.2	105	21	168	NIL	NIL	FPC	
30.	6671	Mahalingam 43/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	12.10.12 To 9.11.12	BT	10200	57	38	5	14	22	13	87	12	159	NIL	NIL	FPC	Good
					AT	10100	57	38	5	14	22	13	88	19	160	NIL	NIL	NIL	

CLINICAL STUDY ON CHARAPARPAM IN – OUT PATIENTS DEPT IN THE MANAGEMENT OF KAMALAI

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD										Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb Gm	Sgr mg/dl	Ur mg/dl	Blood CL	Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
31.	3453	Suganthi 45/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	2.11.12 To 4.11.12	BT	8700	65	32	3	10	15	9	98	22	163	NIL	NIL	NIL	Good
					AT	8400	63	32	5	10	15	10	98	20	162	NIL	NIL	NIL	
32.	3850	Janaki 42/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	4.11.12 To 2.12.12	BT	9400	60	37	3	8	16	11	100	22	159	NIL	NIL	PCS	Good
					AT	9500	60	37	3	8	16	11	98	18	158	NIL	NIL	FPC	
33.	5400	Murugan 38/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	9.11.12 To 7.12.12	BT	9600	65	32	3	10	24	14	88	19	167	NIL	NIL	PCS	Moderate
					AT	9800	65	31	4	10	24	14	89	20	165	NIL	NIL	FPC	
34.	5506	Karthi 40/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	9.11.12 To 7.12.12	BT	8800	58	37	5	14	30	13.5	100	23	147	NIL	NIL	FPC	Good
					AT	8700	55	40	5	14	30	13.5	97	20	149	NIL	NIL	NIL	
35.	4319	Vikesh 32/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	26.12.12 To 2.1.13	BT	9400	67	29	4	17	24	12.9	89	21	158	NIL	NIL	NIL	Good
					AT	9400	67	29	4	17	24	12.9	91	18	164	NIL	NIL	NIL	

SI NO	Op. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION													Results
						BLOOD										Urine			
						TC cells/cumm	DC (%)			ESR(mm)		Hb gm	Sgr mg/dl	Ur mg/dl	Blood CL	Sgr	Alb	Dep	
							P	L	E	½ hr	1 hr								
36	1923	Natraj 18/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes.	25.12.12 To 3.1.13	BT	9300	53	43	4	18	20	14	100	20	173	NIL	NIL	PCS	Good
					AT	9300	53	43	4	15	25	14	96	18	168	NIL	NIL	NIL	
37	2538	Mariyammal 44/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	26.12.12 To 1.1.13	BT	9800	59	37	4	15	24	11	94	24	158	NIL	NIL	FPC	Good
					AT	9800	59	36	5	15	20	11.3	96	22	152	NIL	NIL	NIL	
38	2897	Narasimman 45/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	27.12.12 To 4.1.13	BT	9200	57	39	4	26	44	10.5	130	23	165	NIL	NIL	FPC	Good
					AT	9200	55	40	5	27	43	10.6	117	20	154	NIL	NIL	NIL	
39.	6567	Thamari 25/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	29.12.12 To 5.1.13	BT	8600	61	34	5	10	15	12	95	24	159	NIL	NIL	FPC	Good
					AT	8700	58	39	3	14	20	12	93	18	165	NIL	NIL	NIL	
40	4355	Sudhar 37/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored eyes	30.12.12 To 5.1.13	BT	9100	60	34	6	15	33	10	85	21	158	NIL	NIL	NIL	Moderate
					AT	9100	61	37	2	15	30	10.5	86	19	144	NIL	NIL	NIL	

CLINICAL STUDY ON CHARAPARPAM IN – OUT PATIENTS DEPT IN THE MANAGEMENT OF KAMALAI

SI NO	IP. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION														Results
						BLOOD									Blood CL	Urine			X ray bms/ Endoscopy	
						TC cells/ cumm	DC (%)			ESR(mm)		Hb gm	Sgr mg/ dl	Urm g/ dl		Sgr	Alb	Dep		
							P	L	E	½ hr	1 hr									
1.	550/ 2512	Pandu 55/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	30.9.12 To 19.10.12	BT	9800	60	37	3	15	40	9	106	18	153	NIL	NIL	PCS	-	Poor
					AT	9700	60	38	2	10	48	9.5	102	20	159	NIL	NIL	NIL		
2.	575/ 5202	Varadhan 50/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	7.10.12 To 28.10.12	BT	9600	57	41	2	18	25	10.5	114	23	155	NIL	NIL	PCS	-	Good
					AT	9700	57	41	2	11	33	11	108	20	150	NIL	NIL	NIL		
3.	744/ 1449	Rajamanikam 58/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	30.10.12To 13.11.12	BT	10200	62	34	4	9	19	10	106	19	170	NIL	NIL	FPC	-	Satisfactory
					AT	9800	60	35	5	9	20	10.7	102	23	173	NIL	NIL	NIL		
4.	985/ 485	Selvakumar 65/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	22.11.12 To 9.12.12	BT	9800	60	35	5	6	13	9.6	108	18	169	NIL	NIL	FPC	-	Good
					AT	9100	60	36	4	8	19	10	102	21	163	NIL	NIL	NIL		
5.	948/ 1398	Sagundhala 36/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	24.11.12 To 12.12.12	BT	9400	65	33	2	6	10	9	110	20	166	NIL	NIL	NIL	-	Satisfactory
					AT	9100	63	34	3	4	12	9.6	106	23	160	NIL	NIL	NIL		

CLINICAL STUDY ON CHARAPARPAM IN – OUT PATIENTS DEPT IN THE MANAGEMENT OF KAMALAI

SI NO	IP. No	Name/ Age/ Sex	Complaints	Duration of Days	BT & AT	INVESTIGATION														Results
						BLOOD										Urine			X ray bms/ Endoscopy	
						TC cells/ cumm	DC (%)			ESR(mm)		Hb Gm	Sgr mg/ dl	Urm g/ dl	Blood CL	Sgr	Alb	Dep		
							P	L	E	½ hr	1 hr									
6.	538/ 2112	Palanisami 67/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	30.9.12 To 18.10.12	BT	9800	60	37	3	15	40	9	106	18	153	NIL	NIL	PCS	-	Poor
					AT	9700	60	38	2	10	48	9.5	102	20	159	NIL	NIL	NIL		
7.	537/ 3342	Valarmathi 60/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	8.10.12 To 29.10.12	BT	9600	57	41	2	18	25	10.5	114	23	155	NIL	NIL	PCS	-	Good
					AT	9700	57	41	2	11	33	11	108	20	150	NIL	NIL	NIL		
8.	740/ 1339	Ranjith 58/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	29.10.12 To 19.11.12	BT	10200	62	34	4	9	19	10	106	19	170	NIL	NIL	FPC	-	Satisfactory
					AT	9800	60	35	5	9	20	10.7	102	23	173	NIL	NIL	NIL		
9.	901/ 3390	Saroja 45/Female	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	21.11.12 To 13.12.12	BT	9800	60	35	5	6	13	9.6	108	18	169	NIL	NIL	FPC	-	Good
					AT	9100	60	36	4	8	19	10	102	21	163	NIL	NIL	NIL		
10.	949/ 1988	Manikandan 36/Male	Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera	25.11.12 To 13.12.12	BT	9400	65	33	2	6	10	9	110	20	166	NIL	NIL	NIL	-	Satisfactory
					AT	9100	63	34	3	4	12	9.6	106	23	160	NIL	NIL	NIL		

LIVER FUNCTION TEST

S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dl)	Total protein g/dl	Albumin g/dl
1.	1818	Amudhan 18/Male	24.7.12 To 24.8.12	BT	100	97	133	2.6	4.8	4.1
				AT	41	31	112	1.4	6.1	4.4
2.	3436	Babu 44/Male	1.8.12 To 31.8.12	BT	40	45	77	2.5	4.5	2.8
				AT	19	26	68	1.7	4.6	2.8
3.	2857	Kalaivani 45/Female	2.8.12 To 30.8.12	BT	57	62	95	3.2	6.3	3.2
				AT	35	27	93	1.2	7.1	4.1
4.	6798	SathiyaThilaga 25/Female	8.8.12 To 5.9.12	BT	60	58	74	2.8	6.8	5.1
				AT	24	38	71	1.3	7.6	5.3
5.	4673	Vasanth 37/male	8.8.12 To 4.9.12	BT	65	43	85	3.4	5.1	2.7
				AT	32	25	69	1.2	6.1	3.6

S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dl)	Total protein g/dl	Albumin g/dl
6.	4534	Seetharam 46/Male	9.8.12 To 7.9.12	BT	68	55	79	2.8	4.1	2.5
				AT	22	20	74	1.4	4.6	2.9
7.	7089	Mahendran 27/Male	16.8.12 To 14.9.12	BT	63	58	86	2.6	6.1	4.1
				AT	27	24	70	1.2	7.1	4.5
8.	6897	Sudha 56/Female	16.8.12 To 12.9.12	BT	76	64	98	1.3	6.1	3.5
				AT	24	25	91	0.8	7.3	3.8
9.	5768	Maran 22/Male	18.8.12 to 16.9.12	BT	58	53	75	3.8	5.1	2.7
				AT	37	36	72	1.4	6.2	3.7
10.	8609	David 40/Male	20.8.12 To 17.9.12	BT	56	50	85	2.5	7.2	4.4
				AT	34	41	81	0.8	7.5	4.9

LIVER FUNTION TEST

S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dl)	Total protein g/dl	Albumin g/dl
11.	8978	Ragu 28/Male	22.8.12 To 19.9.12	BT	56.5	65	124	2.1	5.2	3.8
				AT	30	36	109	1.6	5.4	4.9
12.	9032	Ravi 40/male	22.8.12 To 21.9.12	BT	56	53	82	2.5	5.1	2.8
				AT	28	21	65	1.2	6.4	3.9
13.	9098	Madhavan 52/male	22.8.12 To 19.9.12	BT	57	44	97	2.4	6.1	3.1
				AT	54	39	95	1.0	6.4	3.4
14.	253	Kumar 22/male	25.8.12 To 21.9.12	BT	88	70	98	2.8	5.4	3.7
				AT	45	31	91	1.2	6.4	4.0
15.	162	Lakshmi 45/female	25.8.12 To 21.9.12	BT	56	48	102	2.8	6.3	3.5
				AT	43	31	97	1.2	7.1	4.9

LIVER FUNTION TEST

S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dl)	Total protein g/dl	Albumin g/dl
16.	294	Vaithilingam 49/Male	25.8.12 To 21.9.12	BT	56	46	95	3.2	6.3	4.3
				AT	24	19	91	1.3	5.7	4.8
17.	355	Visalatchi 42/Female	26.8.12 To 23.9.12	BT	130	72	102	1.3	6.4	3.4
				AT	28	26	97	0.8	6.8	4.2
18.	405	Kumarai 35/Female	26.8.12 To 24.9.12	BT	78	60	122	1.6	6.4	3.6
				AT	36	40	101	1.0	6.9	3.8
19.	776	Santhosh 26/Male	26.8.12 To 23.9.12	BT	440	480	170	1.5	7.2	4.1
				AT	55	41	120	1.1	6.5	4.9
20.	2457	Mohamed Mustafa 51/male	2.9.12 To 28.9.12	BT	260	220	120	1.9	7.8	3.9
				AT	55	48	110	0.8	7.3	3.6

LIVER FUNTION TEST

S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dl)	Total protein g/dl	Albumin g/dl
21.	3001	Thangaraj 29/Male	5.9.12 To 3.10.12	BT	325	166	106	1.8	8.2	5.3
				AT	42	34	99	1.3	8.1	5.0
22.	4347	Dhandapani 40/Male	8.9.12 To 5.10.12	BT	428	216	124	1.6	5.8	3.2
				AT	34	45	110	1.1	5.4	3.0
23.	5675	Ravisankar 45/Male	17.9.12 To 15.10.12	BT	228	197	110	1.9	6.4	4.5
				AT	26	27	98	0.8	6.3	4.5
24.	5674	Vinoth 55/Male	22.9.12 To 19.10.12	BT	280	97	114	1.6	5.8	3.3
				AT	36	26	100	1.1	6.4	4.2
25.	9555	Punitha 44/Female	22.9.12 To 21.10.12	BT	97	52	138	1.8	5.6	3.6
				AT	33	22	103	1.1	5.2	3.4

LIVER FUNTION TEST

S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dl)	Total protein g/dl	Albumin g/dl
26.	2198	Rajapriyan 33/Male	29.9.12 To 26.10.12	BT	285	98	143	4.8	7.3	4.6
				AT	52	42	110	1.6	7.1	4.1
27.	3651	Geethadevi 28/Female	3.10.12 To 31.10.12	BT	165	87	118	2.1	6.5	3.3
				AT	30	33	96	1.1	6.8	3.7
28.	5707	Saravanan 24/male	10.10.12 To 6.11.12	BT	560	610	180	1.6	6.2	3.4
				AT	36	44	115	0.9	6.4	3.6
29.	3897	Deepak 31/.Male	4.10.12 To 2.11.12	BT	428	512	164	1.7	6.6	4.5
				AT	48	52	110	1.1	6.8	4.1
30.	6671	Mahalingan 43/Male	12.10.12 To 9.11.12	BT	587	494	130	1.9	6.3	2.7
				AT	48	39	99	0.8	5.9	3.4

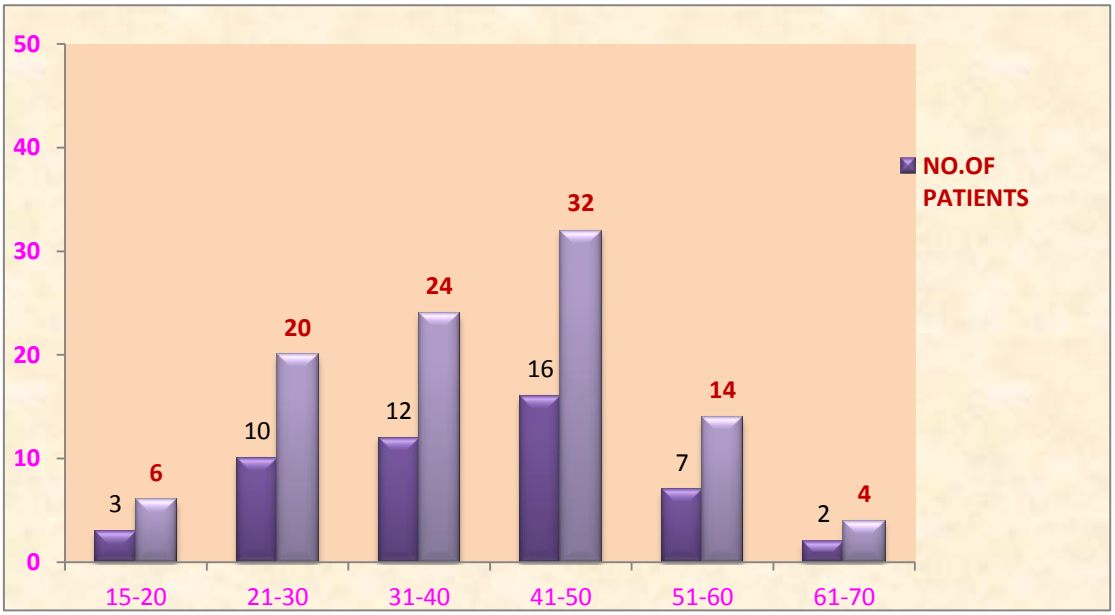
S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dl)	Total protein g/dl	Albumin g/dl
31	3453	Suganthi 45/Female	2.11.12 To 4.11.12	BT	120	72	102	1.3	6.4	3.4
				AT	28	26	97	0.8	6.8	4.2
32	3850	Janaki 42/Female	4.11.12 To 2.12.12	BT	85	78	114	1.6	6.8	4.5
				AT	21	24	98	1.1	7.1	4.8
33	5400	Murugan 38/Male	9.11.12 To 7.12.12	BT	766	685	108	2.8	7.5	4.7
				AT	172	154	96	1.4	7.8	5.2
34	5506	Karthi 40/Male	9.11.12 To 7.12.12	BT	675	560	140	1.6	5.2	4.3
				AT	165	139	102	1.1	5.8	4.8
35	4319	Vikesh 32/Male	26.12.12 To 2.1.13	BT	724	674	130	1.8	7.3	5.1
				AT	120	136	112	1.4	7.1	5.4

S. No	OP No	Name/age/sex	Duration of Treatment	Duration	LIVER FUNCTION TEST					
					AST(SGOT) (IU/L)	ALT(SGPT) (IU/L)	ALP (IU/L)	Total Bilirubin	Total protein	Albumin g/dl
36	1923	Natraj 18/Male	25.12.12 To 3.1.13	BT	536	496	125	1.8	7.6	3.4
				AT	112	120	102	1.2	7.4	3.8
37	2538	Mariyammal 44/Female	26.12.12 To 1.1.13	BT	182	96	98	1.3	6.7	3.8
				AT	34	25	93	0.9	6.9	3.9
38	2897	Narasimman 45/Male	27.12.12 To 4.1.13	BT	658	592	124	1.4	6.4	4.3
				AT	129	131	106	1.1	6.9	4.8
39	6567	Thamari 25/Female	29.12.12 To 5.1.13	BT	46	52	125	1.4	6.7	3.9
				AT	18	22	110	0.8	6.9	4.0
40	4355	Sudhar 37/Male	30.12.12 To 5.1.13	BT	482	283	120	1.7	5.8	2.8
				AT	98	34	99	0.8	6.7	3.4

AGE WISE DISTRIBUTION

SL. NO	AGE IN YEARS	NO. OF PATIENTS	PERCENTAGE (%)
1	15-20	3	6
2	21-30	10	20
3	31-40	12	24
4	41-50	16	32
5	51-60	7	14
6	61-70	2	4
TOTAL		50	100

AGE WISE DISTRIBUTION

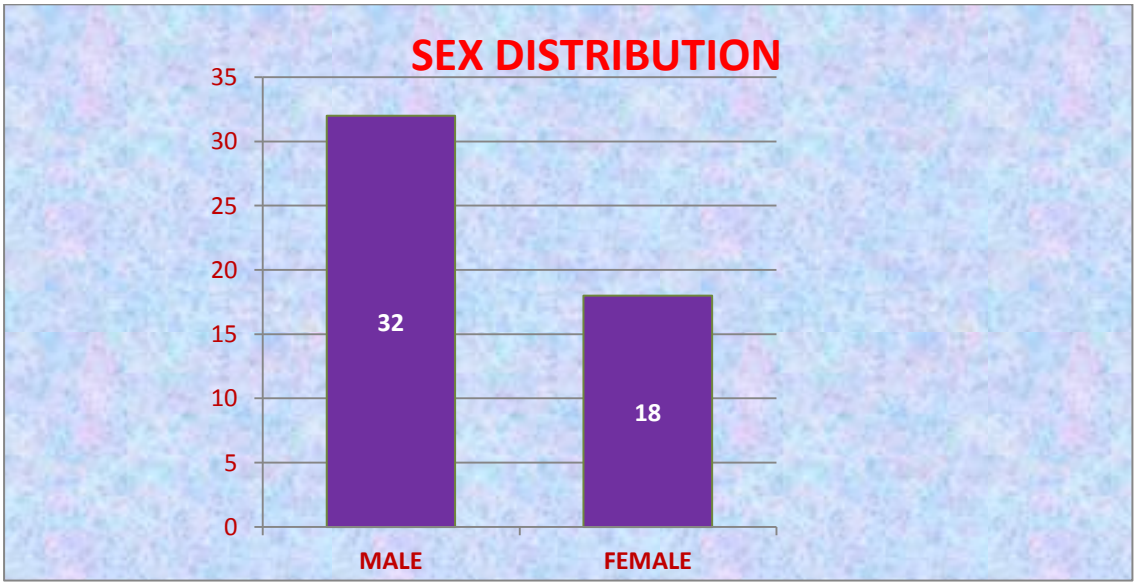


INFERENCE: Among 50 patients,

- 3 patient belongs to the age group of 15-20 years
- 10 patient belongs to the age group of 21-30 years
- 12 patients belongs to the age group of 31-40 years
- 16 patients belongs to the age group of 41-50 years
- 7 patients belongs to the age group of 51-60 years
- 2 patients belongs to the age group of 61-70 years

SEX DISTRIBUTION

SL. NO	SEX	NO. OF PATIENTS	PERCENTAGE
1	Male	32	64
2	Female	18	36
TOTAL		50	100



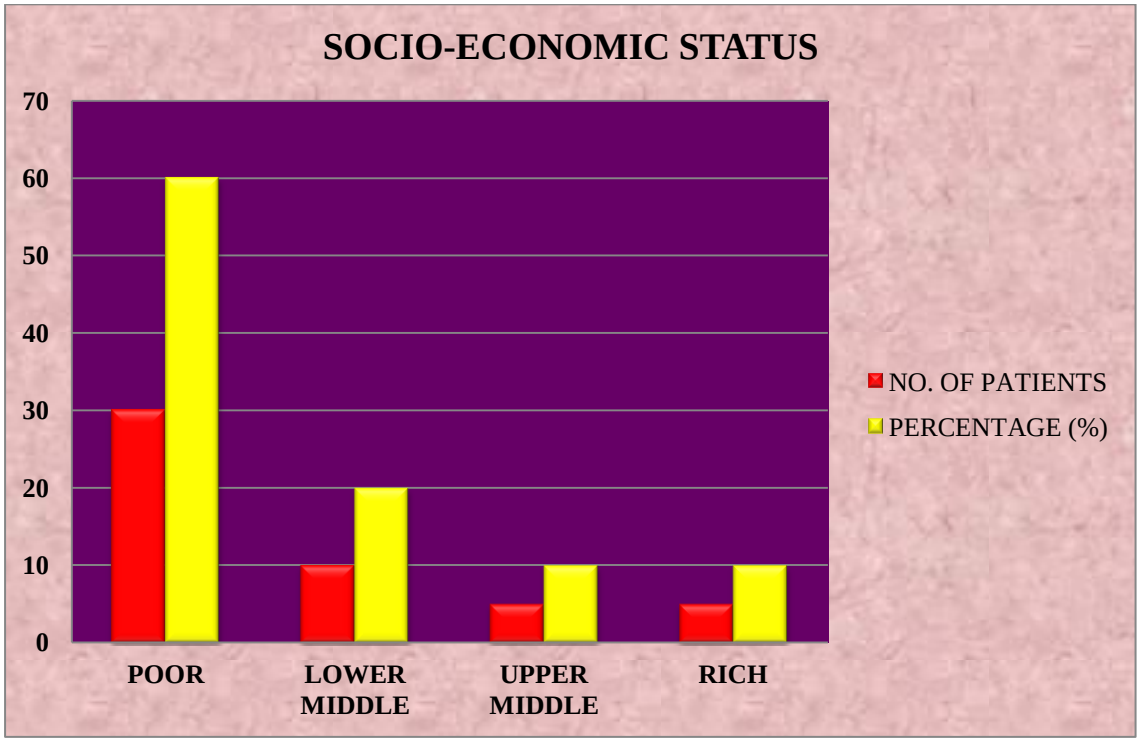
INFERENCE:

Among 50 patients,

- 24 patients were Male
- 26 patients were Female

SOCIO-ECONOMIC STATUS

SL. NO	SOCIO – ECONOMIC STATUS	NO. OF PATIENTS	PERCENTAGE (%)
1	Poor	30	60
2	Lower middle	10	20
3	Upper middle	5	10
4	Rich	5	10
TOTAL		50	100



INFERENCE:

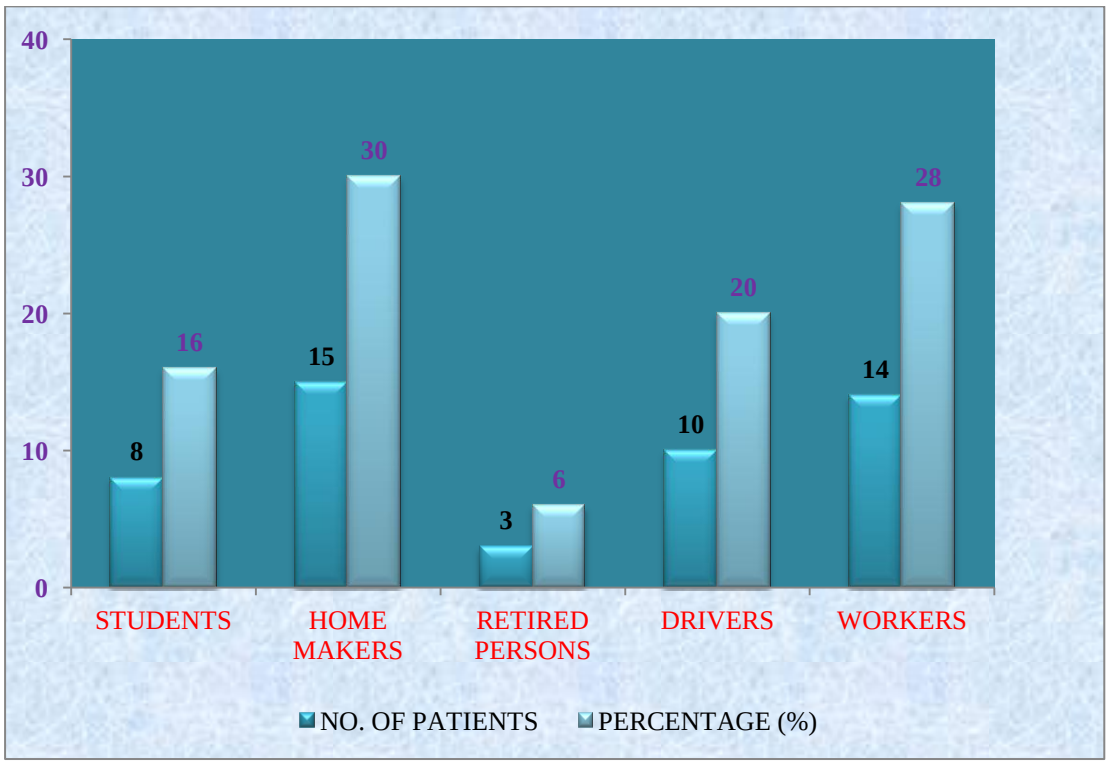
Among 50 patients,

- 30 patients were poor.
- 10 patients were lower-middle.
- 5 patients were upper middle.
- 5 patients were rich.

OCCUPATIONAL STATUS

SL. NO	OCCUPATION	NO. OF PATIENTS	PERCENTAGE (%)
1	Students	8	16
2	Home makers	15	30
3	Retired persons	3	6
4	Drivers	10	20
5	Workers	14	28
TOTAL		50	100

OCCUPATIONAL STATUS

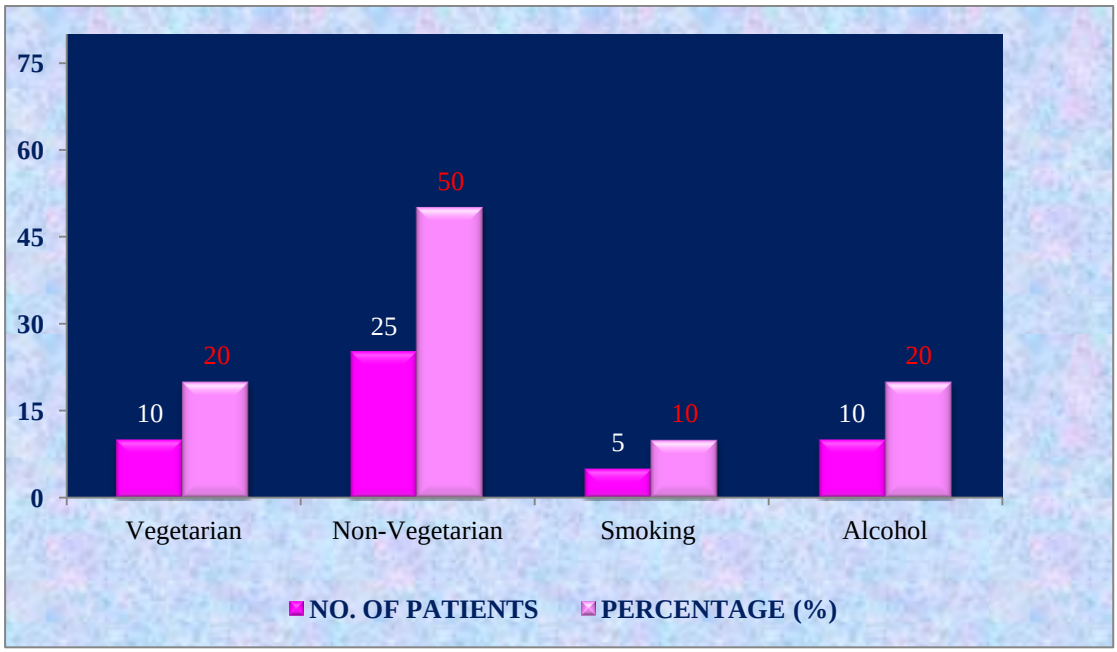


INFERENCE: Among 50 patients,

- 8 patients were Students.
- 15 patients were Home makers.
- 3 patients were Retired persons.
- 10 patients were Drivers.
- 14 Workers

PERSONAL HABITS

SL. NO	PERSONAL HABITS	NO. OF PATIENTS	PERCENTAGE (%)
1	Vegetarian	10	20
2	Non-vegetarian	25	50
3	Smoking	5	10
4	Alcohol	10	20



5. RESULTS AND DISCUSSION:

5.1.PHYSICOCHEMICAL PROPERTY OF CHARA PARPAM:

Table.21

S.No	Parameter	Mean Value
1.	Loss on Drying at 105°C	0.7 %
2.	Total Ash	98.9 %
3.	Acid insoluble Ash	4.5 %
4.	Particle size	Completely passes through sieve no.44
5.	pH	7.5

5.2.FOURIER TRANSFORM INFRARED SPECTROSCOPY (FTIR)

Table.22. FTIR RESULTS of *CHARA PARPAM*

CHARACTERISTIC	FUNTIONAL GROUPS
3853	Amide (N- H stretch)
3430	Alcohol/ Phenol O-H stretch
2925	Carboxylic Acid O-H stretch
2139	Nitrile C=N stretch
1638	Alkenyl C=C stretch
1396	Aromatic C=C Bending
1161	Alkenes C=H stretch
798	Aromatic C=H Bending

Figure.17. SCANNING ELECTRON MICROSCOPE (SEM):

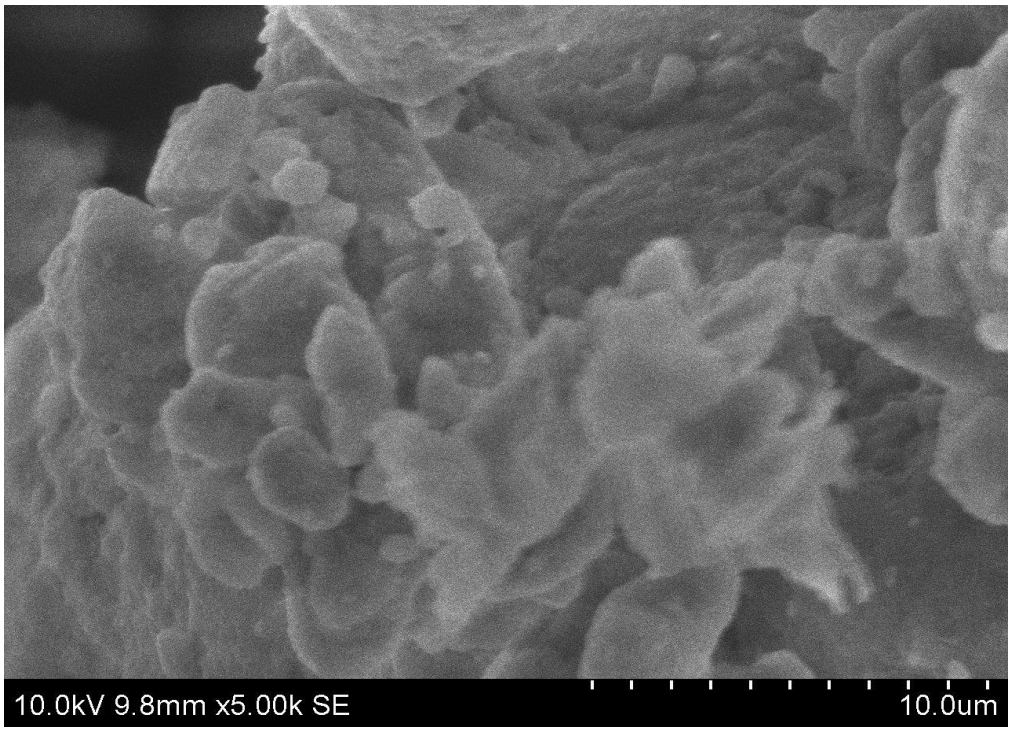
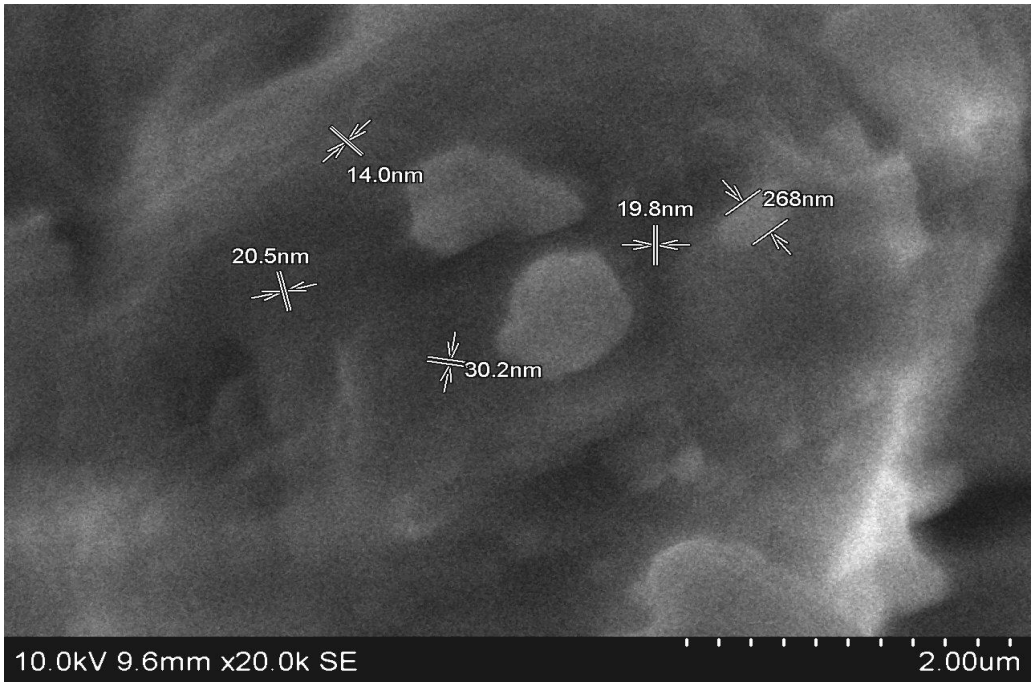


Figure.18



Results:

SEM picture shows Nano particle (Micro level 14.0 nm) size of the sample.

Physical properties of known elements and materials can change as their surface to area ratio is dramatically increased, i.e. when nano scale sizes are achieved. These changes do not take place when going from macro to micro scale. Changes in physical properties such as colloidal properties, solubility and catalytic capacity have been found very useful in areas of bioremediation and drug delivery. The extremely small size of nano particles allows them to penetrate cells and interact with cellular molecules. Due to nano particle size a low dose of the drug can cure the diseases.

5.4. BIO – CHEMICAL ANALYSIS OF CHARA PARPAM:

Results:

The bio – chemical analysis of Chara Parpamshowed the following chemicals,

Sulphate, Chloride, Iron,Calcium, Potassium.

5.5.TOXICOLOGICAL STUDY:

Acute and sub acute toxicity study on Chara parpam in Rodents:

The results of haematological investigations conducted on day 28, revealed following significant changes in the values of different parameters investigated when compared with those of respective controls; However, the increase or decrease in the values obtained was within normal biological and laboratory limits. Biochemical investigations revealed significant changes in the values of different parameters studied when compared with those of respective controls; The animals from control survived throughout the dosing period of 28 days but the 10 mg/kg chara parpam treated dose group animals were shown toxic symptoms and mortality on 13^{th} day after treatment. Signs of major or significant intoxication were observed in animals in higher dose groups during the dosing period of 28 days.

Animals from all the treated dose groups exhibited comparable body weight gain in the second half duration with that of control. Food consumption of control and treated animals was found to be comparable throughout the dosing period of 28days. Ophthalmoscopic examination, conducted prior to and at the end of dosing period on

animals from control and all the treated dose groups did not reveal any significant abnormality except liver enlargement. Functional observation tests conducted at termination revealed no abnormalities. Urine analysis, conducted at the end of the dosing period in week 4 and at the end of recovery period in week 6, revealed no abnormality attributable to the treatment. Organ weight data of animals sacrificed at the end of the dosing period was found to be comparable with that of respective controls. Histopathological examination revealed abnormality in kidney, lung and brain.

Table 23: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose mg/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	50	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2	100	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3	250	+	+	-	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	+	+
4	500	+	+	-	+	-	+	+	-	-	+	-	-	+	-	-	+	-	+	+	+
5	1000	+	+	-	+	-	+	+	-	+	+	-	-	+	+	-	+	-	+	+	+
6	2000	+	+	-	+	-	+	+	-	+	+	-	-	+	+	-	+	-	+	+	+

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality.

Table 24.The effects of the Chara Parpam on weight changes in control and treated rats.

Group	Initial Weight	Weeks			
		1	2	3	4
Normal	162.20±11.10	164.11±7.78	172.65±14.74	176.20±0.77	182.24±5.22
2.5mg/kg	168.25±14.00	176.55±14.12	183.15±11.45	185.14±12.30	185.20±10.46
5mg/kg	164.74±13.12	167.32±11.20	175.29±10.25	179.28±11.94	184.34±4.11
10 mg/kg	164.63±8.15	166.42±12.34	174.17±12.11	176.22±11.42	182.52±12.15

Mean ± SEM (n=6) $^{ns}P>0.05$ Vs. Control group.

Table 25.The effects of the Chara Parpam on Kidney, Heart, Liver and Brain in control and treated groups.

Organ	Control	2.5mg/kg	5mg/kg	10mg/kg
Heart (g)	0.42±0.05	0.44±0.05	0.46±0.02	0.48±0.03
Kidney (g)	0.74±0.10	0.76±0.15	0.75±0.24	0.75±0.18
Liver (g)	3.90±0.06	4.22±0.53	4.14±0.50	4.38±0.45
Brain (g)	1.20±0.16	1.25±0.12	1.15±0.15	1.14±0.18

Mean ± SEM (n=6) $^{ns}P>0.05$ Vs. Control group.

Table 26.Effects on of Daily administration of the Chara Parpam for 28days Biochemical Profiles.

Parameter	Control	2.5mg/kg	5mg/kg	10mg/kg
Glucose (mg/dl)	104.61±0.35	105.51±0.42	114.10±0.33	108.28±0.48
Cholesterol(mg/dl)	44.15±0.42	42.27±0.23*	41.50±0.41	44.22±0.34
Triglyceride(mg/dl)	78.72±0.15	85.84±0.12*	86.72±0.14**	88.23±0.32**
HDL (mg/dl)	105.12±0.04	104.8±0.07	113.12±0.07**	111.00±0.08**
LDL (mg/dl)	78.32±2.31	82.40±3.42	92.58±2.35**	94.15±2.26**
Protein (mg/dl)	7.11±0.30	7.82±0.28	7.71±0.32	6.99±0.27
Albumin	3.10±0.20	3.18±0.22	3.12±0.24	3.56±0.20
Globulin	3.16±0.07	3.15±0.06	3.16±0.08	3.14±0.07
Creatinine (mg/dl)	0.20±0.04	0.27±0.04*	0.24±0.05	0.25±0.04
AST IU/L	54.10±1.12	55.21±2.31	55.10±2.20	56.24±2.14
ALT IU/L	25.42±1.76	26.10±2.28	22.18±2.36	24.42±3.22
ALP IU/L	62.26±3.30	65.12±3.11	62.10±1.54	59.88±2.37

Mean ± SEM (n=6) *P<0.05; **P<0.01 Vs. normal group.

Table 27. Serum Bilirubin (µmol/l) levels of rats treated with Chara Parpam for 28 days.

Group	Total Bilirubin	Direct Bilirubin	Unconjugated Bilirubin
Normal	14.66±2.00	15.51±1.52	4.32±2.02
2.5mg/kg	14.32±1.11	16.42±1.12	4.56±1.20
5mg/kg	15.14±1.14	15.73±0.72	5.34±1.28
10 mg/kg	16.26±1.22	15.17±0.35	10.22±1.62**

Mean ± SEM (n=6) **P<0.01 Vs. Normal group.

Table 28. Renal function Parameter of rats treated with Chara Parpam for 28 days.

Group	Urea(µmol/l)	Creatinine(µg/dl)	Uric acid(mg/dl)
Normal	6.28±0.50	255.11±4.56	3.32±0.06
2.5mg/kg	7.12±0.15*	265.26±4.32	1.95±0.09**
5mg/kg	7.65±0.22*	270.22±4.15*	2.12±0.07*
10 mg/kg	8.78±0.12**	272.30±5.22*	1.35±0.08**

Mean ± SEM (n=6) *P<0.05; **P<0.01 Vs. Normal group.

Table 29. Serum Electrolyte levels (mmol/l) of rats treated with Chara Parpam for 28 days.

Group	Sodium	Potassium	Bicarbonate
Normal	151.22±0.72	6.10±0.55	27.40±0.50
2.5mg/kg	149.00±1.31	5.82±1.12	25.11±0.66
5mg/kg	147.84±1.12	5.76±0.09	24.42±0.50*
10 mg/kg	145.15±0.45**	4.97±0.10*	25.71±1.32

Mean ± SEM (n=6) *P<0.05; **P<0.01 Vs. Normal group.

Table.30. Urine Analysis

Parameters	Control	2.5mg/kg	5mg/kg	10 mg/kg
Colour	Yellow	Yellow	Yellow	Yellow
Transparency	Clear	Slightly turbid	Slightly cloudy	Slightly turbid
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	>8.0	>8.0	>9.0
Protein	Nil	3+	3+	3+
Glucose	Nil	Nil	Nil	Nil
Bilirubin	-ve	-ve	-ve	-ve
Ketones	-ve	+ve	+ve	+ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Abnormal	Abnormal	Abnormal
Pus cells	0-cells/HPF	1-cell/HPF	2-cells/HPF	1-cell/HPF
RBCs	Nil	Nil	0-1cells/HPF	Nil
Epithelial cells	Nil	1-cell/HPF	Nil	1-cell/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil

SUBACUTE TOXICITY OF CHARA PARPAM:

Figure.19.Bone

LOWER DOSE MID DOSE HIGH DOSE

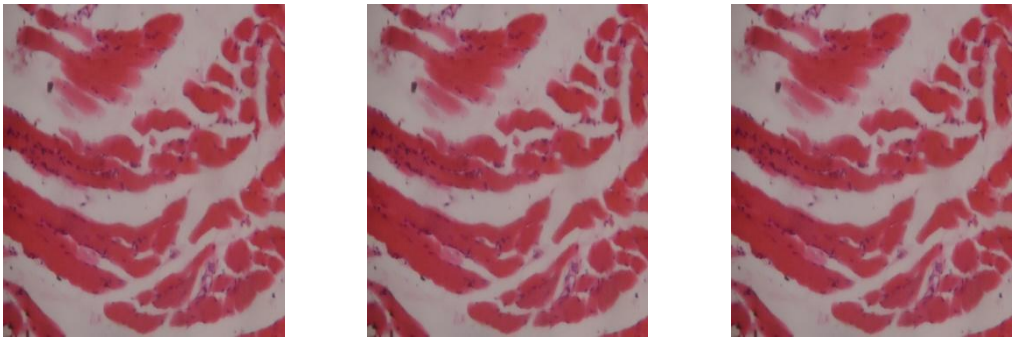


Figure.20.Brain

LOWER DOSE MID DOSE HIGH DOSE

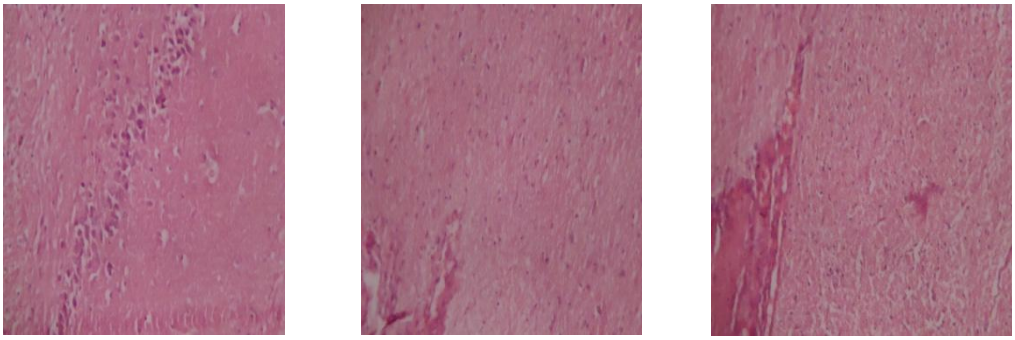
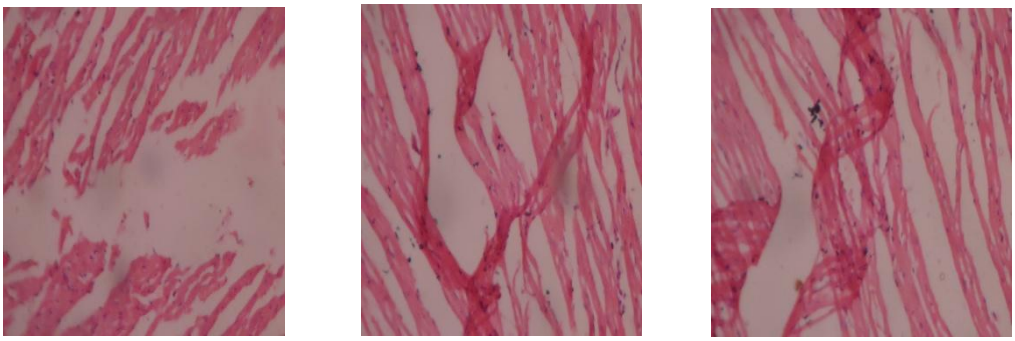


Figure.21.Heart

LOWER DOSE MID DOSE HIGH DOSE



SUBACUTE TOXICITY OF CHARA PARPAM:

Figure.22 Intestine

LOWER DOSE MID DOSE HIGH DOSE

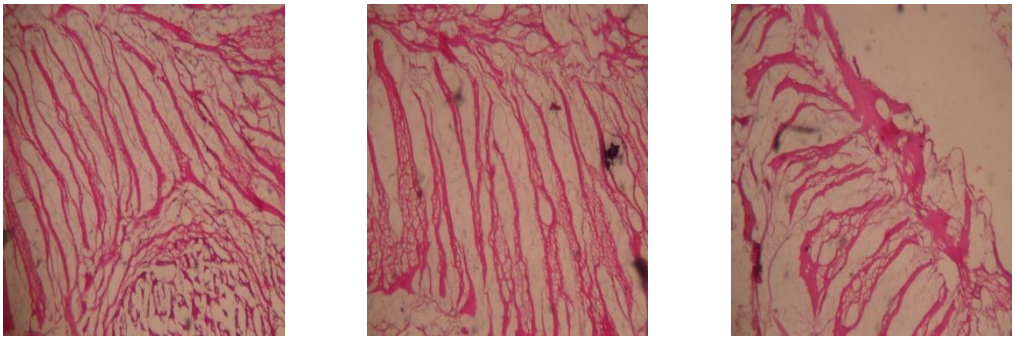


Figure.23.Kidney

LOWER DOSE MID DOSE HIGH DOSE

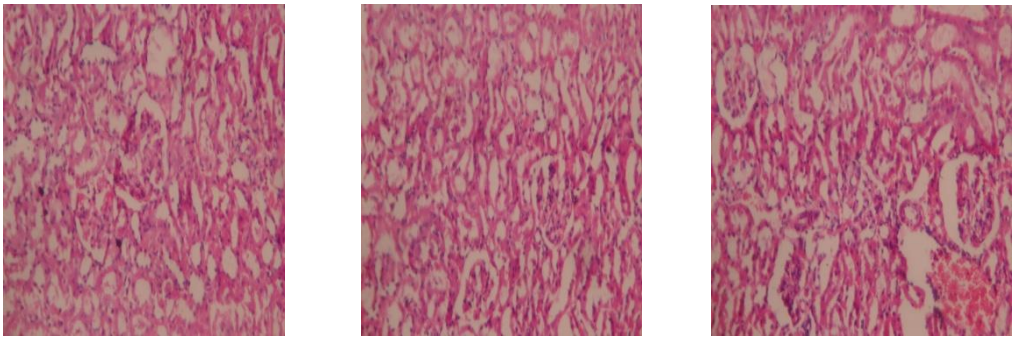
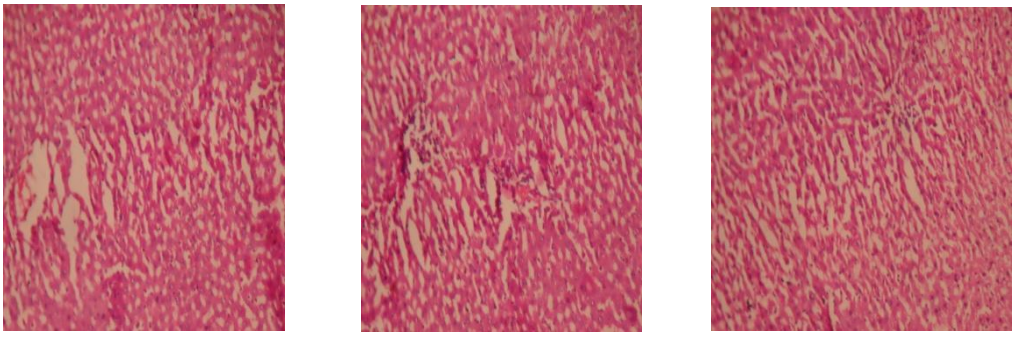


Figure.24. Liver

LOWER DOSE MID DOSE HIGH DOSE



SUBACUTE TOXICITY OF CHARA PARPAM:

Figure.25. Lung

LOWER DOSE MID DOSE HIGH DOSE

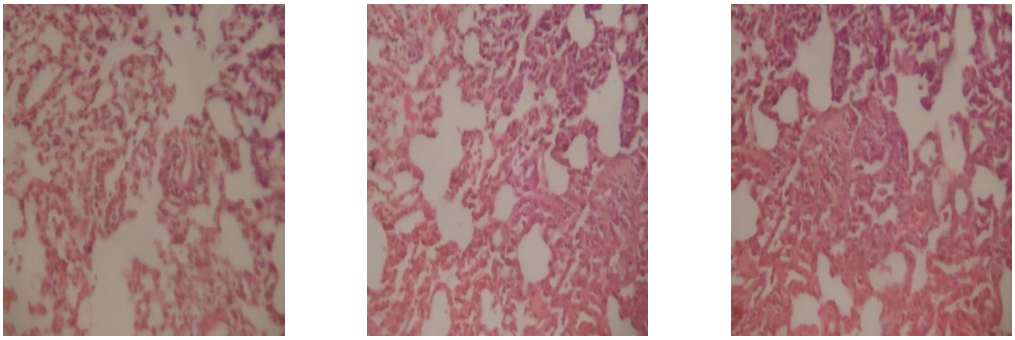


Figure.26. Ovary

LOWER DOSE MID DOSE HIGH DOSE

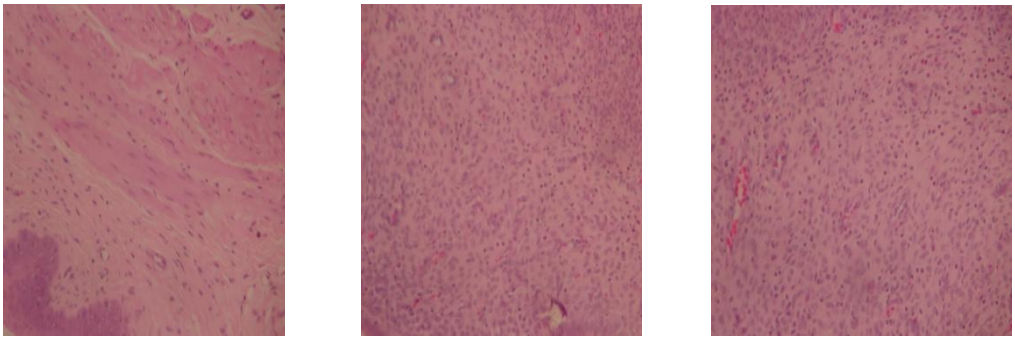
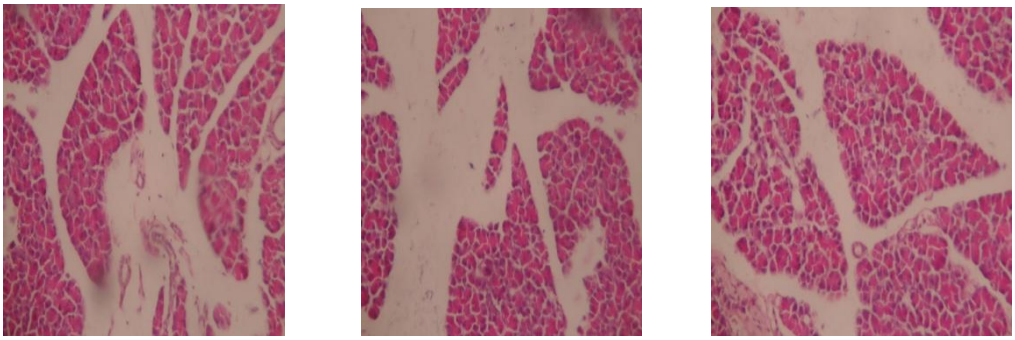


Figure.27.Pancrease

LOWER DOSE MID DOSE HIGH DOSE



SUBACUTE TOXICITY OF CHARA PARPAM:

Figure.28. Spleen

LOWER DOSE MID DOSE HIGH DOSE

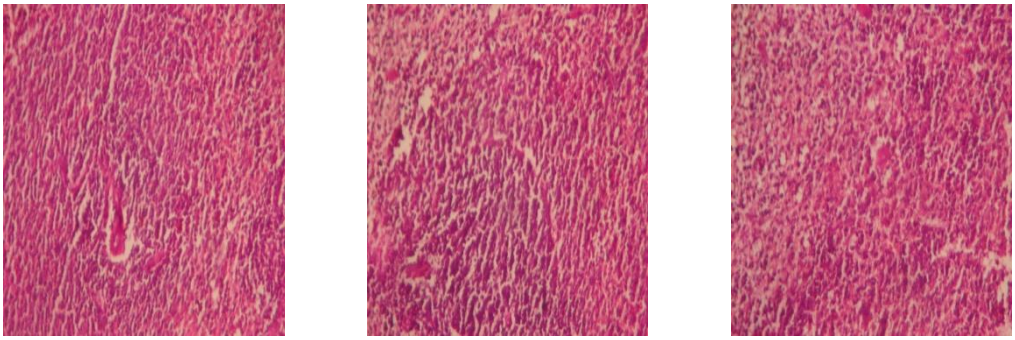


Figure.29. Stomach

LOWER DOSE MID DOSE HIGH DOSE

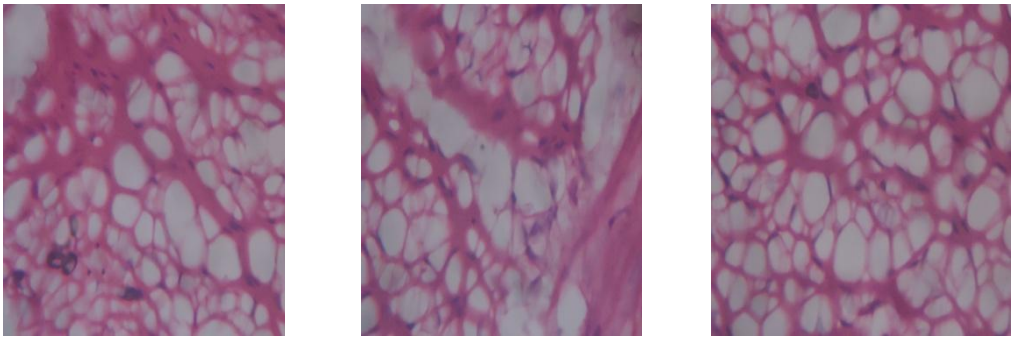
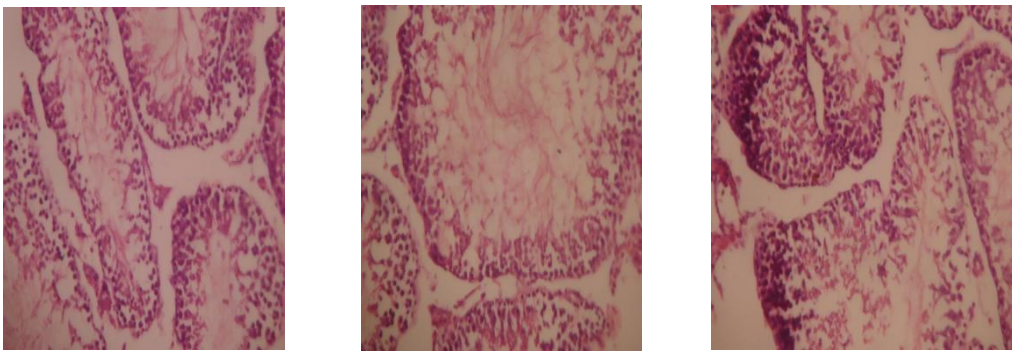


Figure.30. Testis.

LOWER DOSE MID DOSE HIGH DOSE



5.6. PHARMACOLOGICAL ASPECT:

Result and Discussion:

The results of acute oral toxicity study suggested that the Chara Parpam was safe up to 50mg/kg. As per the above study, dose fixation was done and 5mg/kg, and 10mg/kg have been selected as low and high doses respectively for the hepatoprotective study. Administration of Chara Parpam has shown variations in various biochemical parameters in animals induced liver damage by CCL_4 as there was significant increase in liver weight due to damage caused by administration of CCL_4 in control animals as compared to normal group. In animals treated with Silymarin and Chara Parpam, there was significant reduction in liver weight compared to toxic group. Histopathological study of the liver from different groups of rats corroborated the hepatoprotective efficacy of Chara Parpam. Various pathological changes like steatosis, centrilobular necrosis and vacuolization seen in group II (toxicant rats) is due to oxidative damage by free radical generation. These pathological changes were prevented to moderate extent in both test groups.

CCL_4 administration caused significant elevation of ALT, AST,ALP, direct bilirubin and total bilurubin by inducing hepatic damage in control animals as compared to normal animals while, standard drug silymarin treatment reduced concentration in animals of standard group and those were almost equivalent to normal. There was dose dependent significant reduction in serum ALT levels in Chara Parpam treated animals as compared to control group. In positive control group animals treated with CCL_4, there was significant decrease in serum total protein, albumin and serum ionic concentration due to liver damage when compared to saline alone treated animals but administration of silymarin caused significant rise in total protein compared to control group. In animals administered with Chara Parpam there was significant increase total protein level and it was dose dependent. Histological profile of the control animals showed normal hepatocytes.

In toxicant administered animal liver exhibited intense centrilobular necrosis, vacuolization and macrovesicular fatty change. The sections of liver from the standard drug silymarin treated showed hepatic architecture, which was similar to that of normal. The animals treated with Chara Parpam exhibited significant liver protection against the toxicant as evident by presence of normal hepatic cords, absence of necrosis and lesser fatty infiltration. The administration of CCL_4 was resulted into

complete loss of the normal architecture of livers in positive control animals with the appearance of vacuolated hepatocytes and degenerated nuclei. Vacuolization, fatty changes and necrosis of hepatocytes were severe in the centrilobular region. The toxic metabolites of CCL_4 caused excessive formation and deposition of connective tissue and development of scars. The nodular transformation of liver architecture was revealed of liver in the liver sections of rats treated with Chara Parpam 5mg/kg with loss of structure of hepatic lobules. But liver sections treated with high doses of Chara Parpam showed more or less normal lobular pattern with tiny and a mild degree of fatty change, necrosis and lymphocyte infiltration almost comparable to the control and silymarin treated groups. Fatty change or fatty liver is the main consequence of liver toxicity which is characterized by the deposition of fat in liver. Due to the deposition of fat and triglycerides, total weight of liver increases in drug induced hepatic damage. In positive control group, there was significant rise in liver weights but silymarin and Chara Parpam could able to normalize weight of livers in therapeutic groups by decreasing toxicity of liver. Liver is important storage organ and stores various serum enzymes like ALT and AST which are involved in transamination reactions for various amino acids. Alkaline phosphatise is isoenzyme synthesized mainly by liver and has vital role in dephosphorylation of various biomolecules. In liver disease, liver cannot store ALT and AST and increased synthesis of ALP observed due to liver parenchymal damage caused by liver toxicity. Among various, one of the important functions of liver is detoxification of bilirubin which is breakdown product of haem. The bilirubin uptake by liver parenchyma cells from the blood and conjugates with glucoronic acid in presence of enzyme glucuronyl transferase, later congjugated product excreted into bile.

In liver toxicity total bilirubin and direct bilirubin concentration in serum increases due to abnormality in hepatic parenchymal cells. In our present study, CCL_4 elevated levels of ALT, AST, ALP, direct bilirubin and total bilirubin was observed in control animals which may be due to reduced function of liver due to toxicity. Treatment with silymarin and Chara Parpam significantly reduced concentration of above serum parameters in animals of therapeutic groups which could be due to possible protection given by Chara Parpam. Serum total protein, also called plasma total protein or total protein, is a biochemical test for measuring the total amount of protein in blood plasma or serum. The albumin and globulin are the main components of total protein in the plasma and albumin mainly synthesized by the liver. In CCL_4

induced hepatotoxicity, albumin synthesis will be decreased due to cirrhosis and leads to reduction in total protein. Hence serum albumin and total protein are the two important biomarkers of liver function.

The ascites and edema are the two main complications of liver injury which is due to accumulation of fluids in extravascular sites of the body and hence serum sodium and potassium moves into fluids which finally lead to reduced concentration of these ions in blood. In our present study in control animals treated with CCl_4, there was significant reduction in concentration of serum albumin, total protein, serum sodium and potassium due to toxicity. In therapeutic animals treated with standard drug silymarin and Chara Parpam significant increase in serum albumin, total protein, serum sodium and potassium was observed as compared to toxic group.

Histopathological studies of liver in toxicant group cause fatty changes, granular degeneration and inflammation. Also showed marked reduction in fatty degeneration and necrosis in animals treated with standard drug silymarin and Chara Parpam. It is evident that the Chara Parpam caused regeneration of liver parenchyma cells and treated hepatic cell damage due to CCL_4 toxicity.

HISTOPATHOLOGY OF LIVER

Figure.31

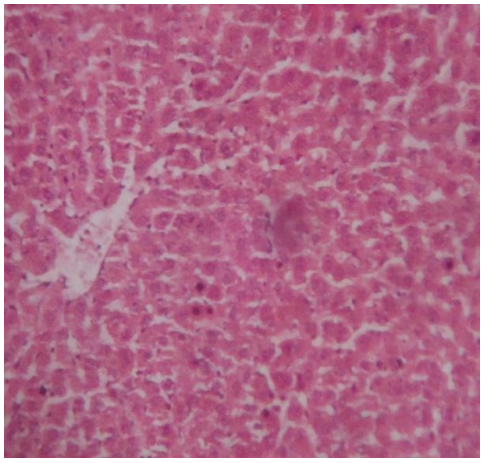


Figure.32

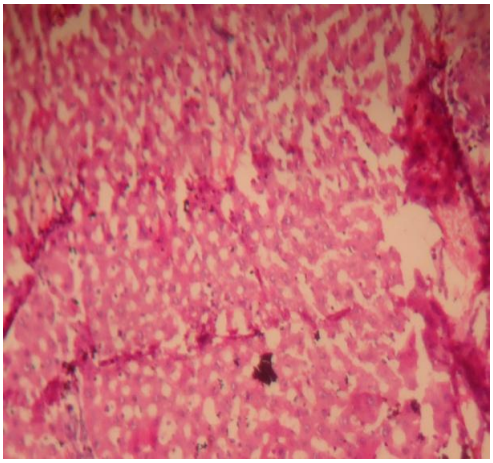


Figure.33.

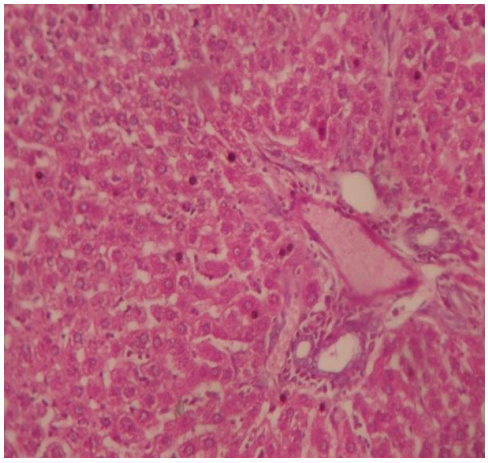


Figure.34.

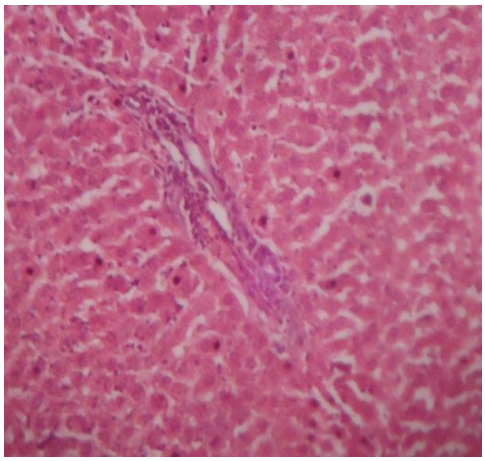


Figure.31. Group I (Normal Liver)

Figure.32. Group II (Control)

Figure.33 Group III (Chara Parpam 5mg)

Figure.34 Group IV (Chara Parpam 10mg)

Table 31: Effect of Chara Parpam on liver weight

GROUP	LIVER WEIGHT (gm)
Normal Saline	5.76± 0.10
Positive Control	8.42±0.68
Chara Parpam 5mg/kg	6.54±0.14*
Chara Parpam 10mg/kg	5.66±0.10**
Silymarin 25mg/kg	4.2±0.04**

Values are mean ± S.E.M, n=6; $^{ns}p>0.05$, *$p<0.05$, **$p<0.01$ Vs positive control.

Table 32:Effect of Chara Parpam on serum biochemical parameters

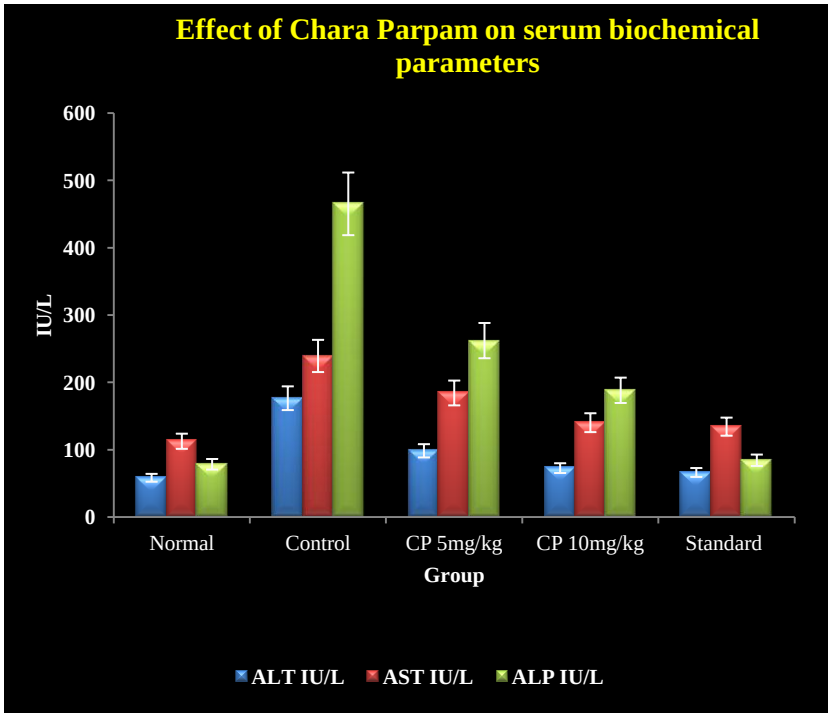
GROUP	ALT IU/L	AST IU/L	ALP IU/L	T. Bilurubin g/l	D. Bilurubin g/dl
Normal Saline	58.21±1.6	112.5±0.52	78.32±1.8	0.36±0.02	0.022±0.010
Positive Control	176.42±26.1	239.2±3.3	465.16±4.05	0.92± 0.02	0.28± 0.03
Chara Parpam 5mg/kg	98.33±2.8**	184.22±1.4**	262.01±2.0**	0.64±0.018**	0.12±0.004**
Chara Parpam 10mg/kg	72.59±2.0**	140.10±2.0**	188.2±1.6**	0.47±0.012**	0.094±0.004***
Silymarin 25mg/kg	66.10±1.2**	134.2±1.4**	84.3±1.65**	0.40±0.01**	0.092±0.02**

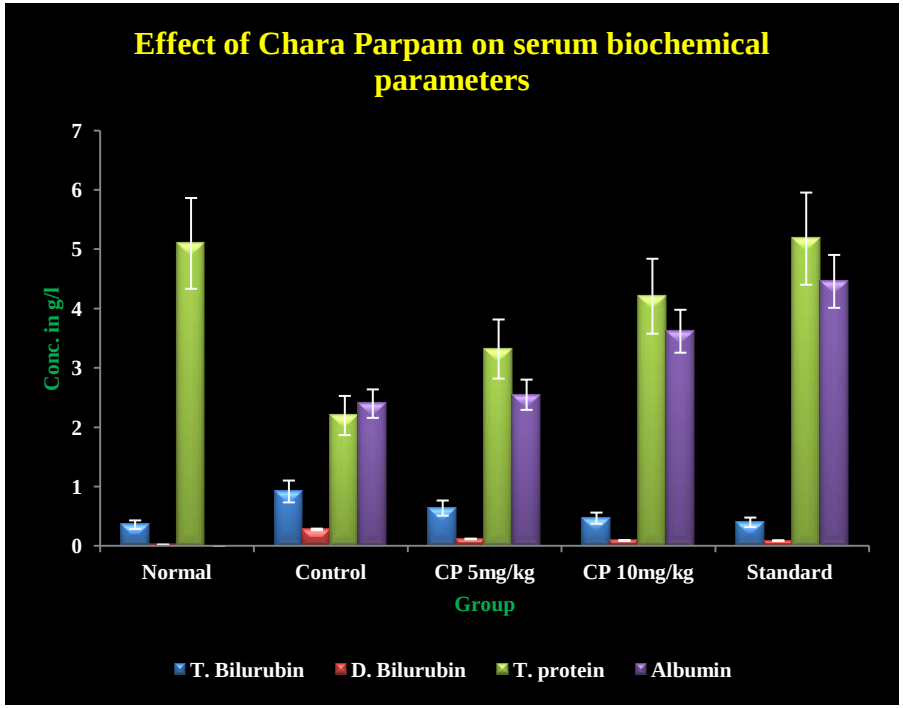
Values are mean ± S.E.M, n=6; $^{ns}p>0.05$, *p<0.05, **p<0.01 Vs control.

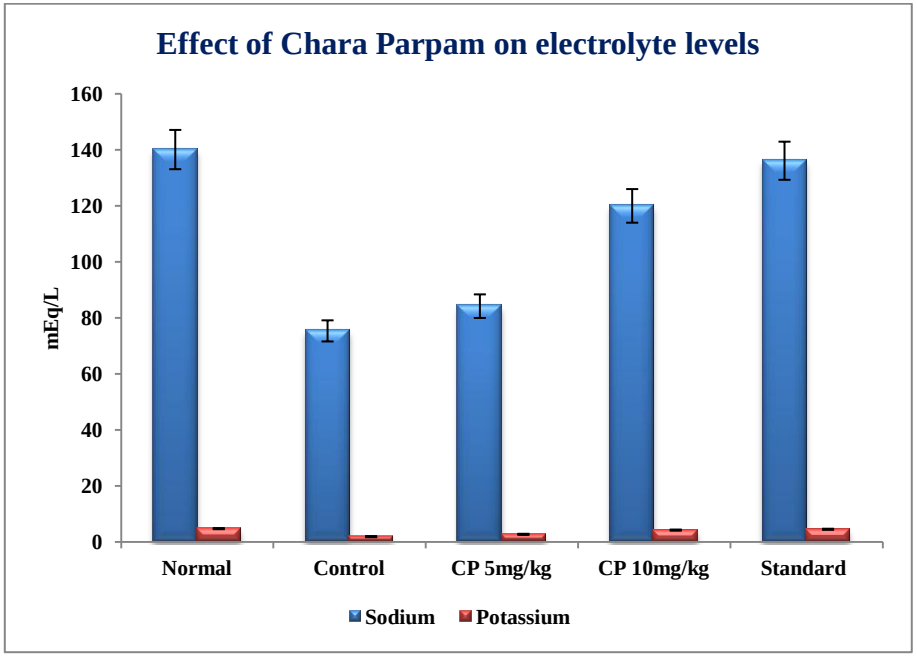
Table 33: Effect of Chara Parpam on serum parameters Total protein, Albumin and ions

GROUP	Total protein g/dl	Albumin g/dl	Sodium mEq/L	Potassium mEq/L
Normal Saline	5.1±0.10	4.2± 0.11	140.12± 0.7	4.85± 0.26
Positive Control	2.2±0.12	2.4± 0.05	75.41± 0.9	2.00± 0.08
Chara Parpam 5mg/kg	3.32± 0.2*	2.55±0.17	84.23± 2.0*	2.81±0.17*
Chara Parpam 10mg/kg	4.21± 0.20**	3.62±0.18**	120.02± 1.4**	4.33±0.16**
Silymarin 25mg/kg	5.18±0.07**	4.46±0.05**	136.13±1.8**	4.62±0.14**

Values are mean ± S.E.M, n=6; $^{ns}p>0.05$, *p<0.05, **p<0.01, Vs positive control.


Effect of Chara Parpam on serum biochemical parameters
600
500
400
300
200
100
0
IU/L
Normal
Control
CP 5mg/kg
CP 10mg/kg
Standard
Group
ALT IU/L
AST IU/L
ALP IU/L


Effect of Chara Parpam on serum biochemical parameters
Conc. in g/l
7
6
5
4
3
2
1
0
Normal
Control
CP 5mg/kg
CP 10mg/kg
Standard
Group
T. Bilurubin
D. Bilurubin
T. protein
Albumin

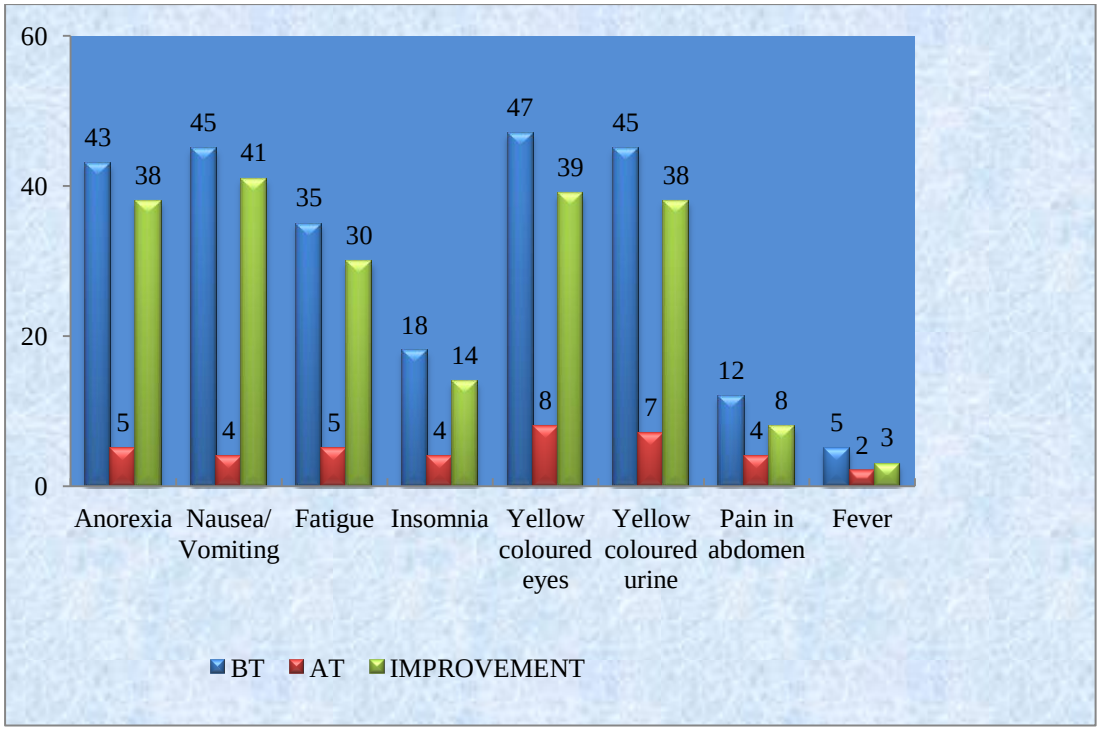

Effect of Chara Parpam on electrolyte levels
160
140
120
100
80
60
40
20
0
mEq/L
Normal
Control
CP 5mg/kg
CP 10mg/kg
Standard
Sodium
Potassium

CLINICAL ASSESMENT

Table: 34. IMPROVEMENT IN SIGNS AND SYMPTOMS

SL. NO	SIGNS AND SYMPTOMS	NO. OF PATIENTS			
		BT	AT	IMP.	IMPROVEMENT (%)
1	Anorexia	43	5	38	88
2	Nausea/ Vomiting	45	4	41	91
3	Fatigue	35	5	30	86
4	Insomnia	18	4	14	78
5	Yellow coloured eyes	47	8	39	83
6	Yellow coloured urine	45	7	38	84
7	Pain in abdomen	12	4	8	67
8	Fever	5	2	3	60

IMPROVEMENT IN SIGNS AND SYMPTOMS



INFERENCE:

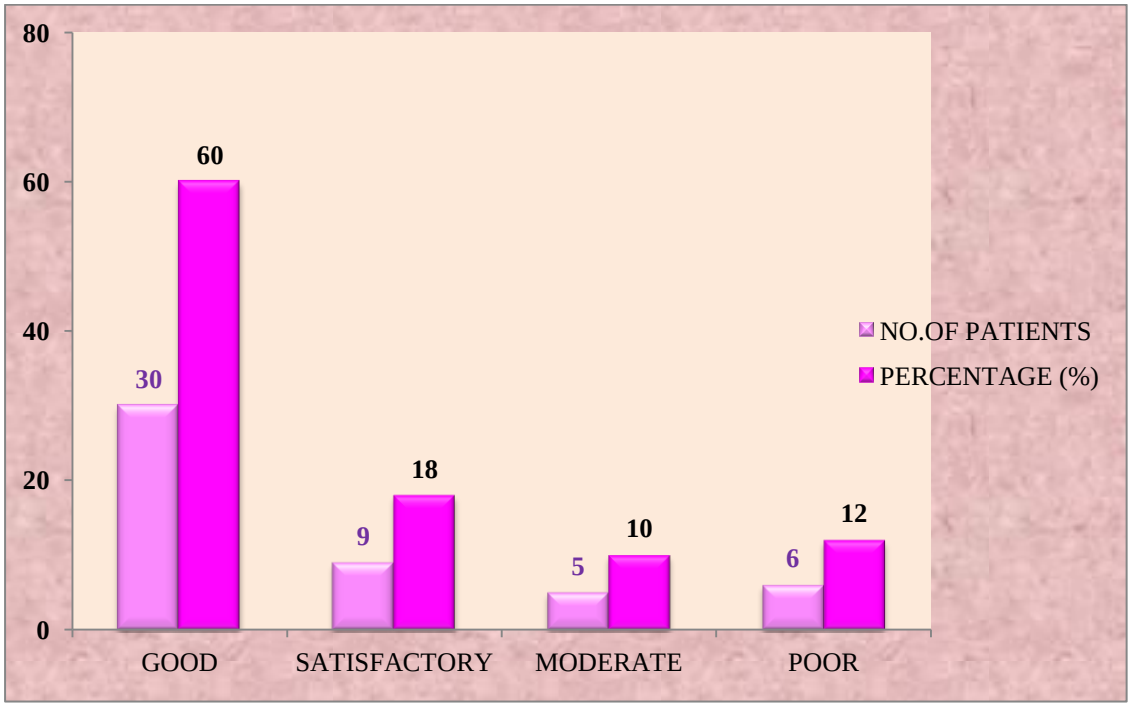
Among 50 patients,

- 38 out of 43 patients were relieved from Anorexia.
- 41 out of 45 patients were relieved from Nausea/ Vomiting.
- 30 out of 35 patients were relieved from Fatigue.
- 14 out of 18 patients were relieved from Insomnia.
- 39 out of 47 patients were relieved from Yellow coloured eyes.
- 38 out of 45 patients were relieved from Yellow coloured urine.
- 8 out of 12 patients were relieved from pain in abdomen.
- 3 out of 5 patients were relieved from Fever.

GRADATION RESULT

SL. NO	LEVEL OF IMPROVEMENT	NO.OF PATIENTS	PERCENTAGE (%)
1	Good	30	60
2	Satisfactory	9	18
3	Moderate	5	10
4	Poor	6	12
TOTAL		50	100

GRADATION RESULT



STATISTICAL ANALYSIS

DESCRIPTIVE STATISTICAL FOR IMPROVEMENT OF LIVER FUNCTION TEST IN *KAMALAI*

PAIRED "t" TEST RESULT:

P value and statistical significance:

AST in Jaundice Patients:

The elevated AST (Aspartate transaminase)/ serum glutamic oxaloacetic transaminase (SGOT) levels were reduced significantly, when compared to the pre-treatment values. The two-tailed P value is less than 0.0001by conventional criteria; this difference is considered to be extremely statistically significant.

Confidence interval:

The mean of Group One minus Group Two equals 30.084

95% confidence interval of this difference: From 23.124 to 38.563

Intermediate values used in calculations:

t = 8.516, df = 15, standard error of difference = 3.622

Treatment	No Patients	Mean	S.D	S.E.M
Before treatment	16	63.281	14.292	3.573
After treatment	16	32.438	9.647	2.412

AST in Alcoholic Liver Disease Patient:

The two-tailed P value is less than 0.0008. By conventional criteria, this difference is considered to be extremely statistically significant.

Confidence interval:

The mean of Group One minus Group Two equals 84.38

95% confidence interval of this difference: From 48.65 to 120.10

Intermediate values used in calculations:

t = 5.58, df = 7 ,standard error of difference = 15.10

Treatment	No Patients	Mean	S.D	S.E.M
Before treatment	8	112.88	45.60	16.12
After treatment	8	28.50	6.28	2.22

AST in Fatty Liver disease:

The two-tailed P value equals 0.0001. By conventional criteria, this difference is considered to be extremely statistically significant.

Confidence interval:

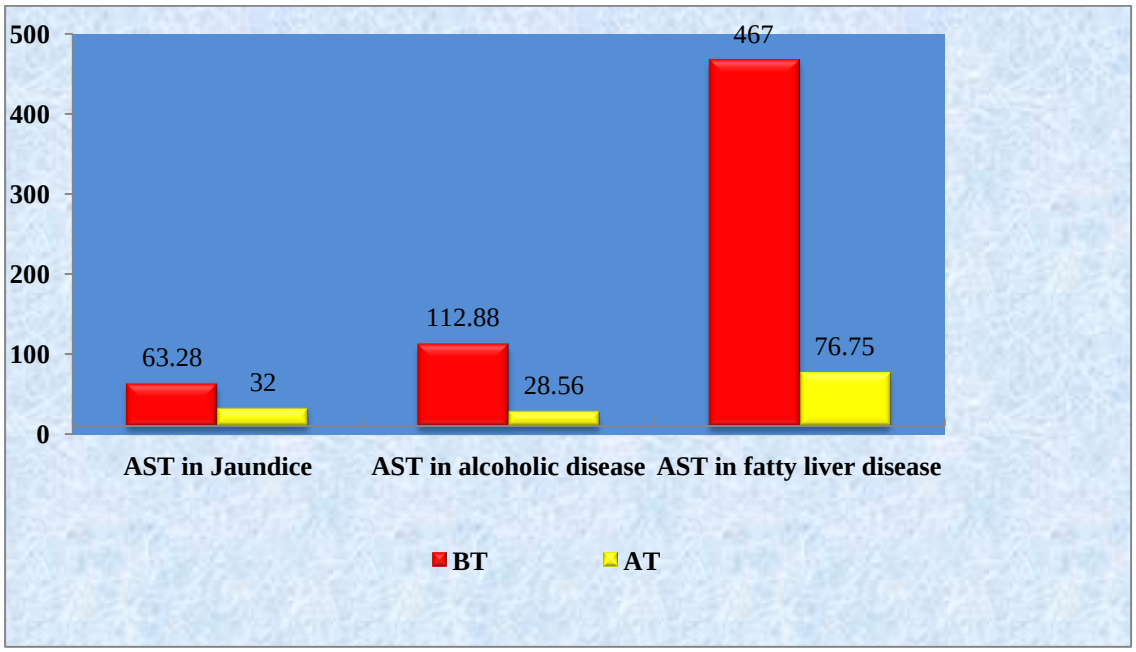
The mean of Group One minus Group Two equals 390.25

95% confidence interval of this difference: From 306.97 to 473.53

Intermediate values used in calculations:

t = 9.98, df = 15, standard error of difference = 39.480

Treatment	No Patients	Mean	S.D	S.E.M
Before treatment	16	467.00	190.66	47.67
After treatment	16	76.75	48.51	12.13



ALT (Alanine transaminase):

The elevated ALT (Alanine transaminase) /serum glutamic pyruvic transaminase (SGPT) levels were also reduced significantly, when compared to the pre-treatment values, The two-tailed P value is less than 0.0001.By conventional criteria, this difference is considered to be extremely statistically significant.

Confidence interval:

The mean of Group One minus Group Two equals 149.00

95% confidence interval of this difference: From 88.71 to 209.29

Intermediate values used in calculations:

t = 5.00,df = 38,standard error of difference = 29.78

Treatment	No Patients	Mean	S.D	S.E.M
Before treatment	40	196.50	212.14	33.54
After treatment	40	45.28	36.41	5.83

ALP: Alkaline Phosphatase:

The elevated alkaline phosphatase levels were also reduced significantly, when compared to the pre-treatment values, the two-tailed P value is less than 0.0001. By conventional criteria, this difference is considered to be extremely statistically significant.

Confidence interval:

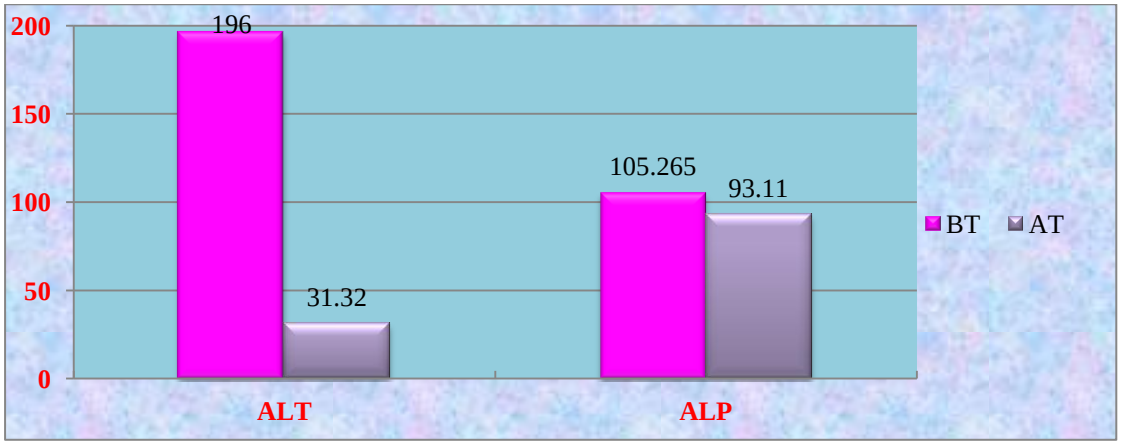
The mean of Group One minus Group Two equals 12.155

95% confidence interval of this difference: From 7.522 to 16.788

Intermediate values used in calculations:

t = 5.3071, df = 39,standard error of difference = 2.290

Treatment	No Patients	Mean	S.D	S.E.M
Before treatment	40	105.265	25.825	4.083
After treatment	40	93.110	15.685	2.480



Bilirubin:

The mean serum bilirubin values were elevated at the time of the enrolment, in all patients in these studies. Cumulative data analysis showed a significant reduction in the mean serum bilirubin level,The two-tailed P value is less than 0.0001By conventional criteria, this difference is considered to be extremely statistically significant.

Confidence interval:

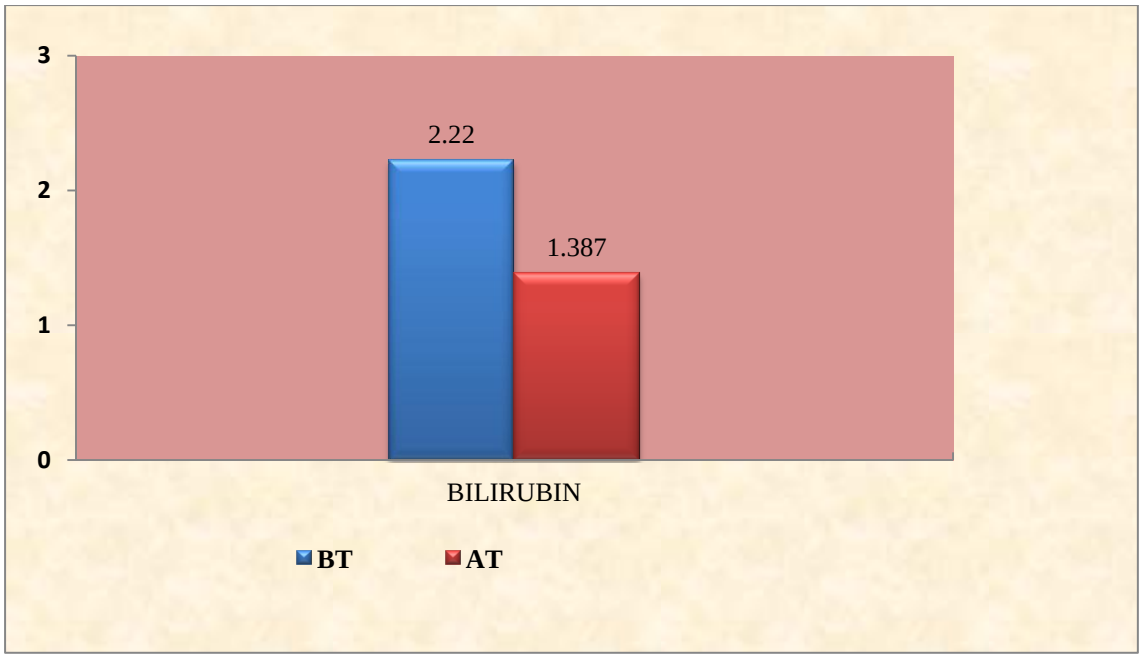
The mean of Group One minus Group Two equals 0.832

95% confidence interval of this difference: From 0.589 to 1.076

Intermediate values used in calculations:

t = 6.003,df = 39, standard error of difference = 0.121

Treatment	No Patients	Mean	S.D	S.E.M
Before treatment	40	2.220	0.887	0.140
After treatment	40	1.387	0.940	0.149



STATISTICAL ANALYSIS

DESCRIPTIVE STATISTICAL FOR IMPROVEMENT OF SIGNS & SYMPTOMS IN *"KAMALAI"*

PAIRED "t" TEST RESULT:

"**p" value & statistical significance:**

Group	N	Mean	SD	SEM
Group I	8	31.25	16.96	6.00
Group II	8	26.38	15.56	5.50

P value and statistical significance:

The two-tailed P value equals 0.0002

By conventional criteria, this difference is considered to be extremely statistically significant.

Confidence interval:

The mean of Group One minus Group Two equals 4.88

95% confidence interval of this difference: From 3.30 to 6.45

Intermediate values used in calculations:

t = 7.3145

df = 7

standard error of difference = 0.666

DISCUSSION:

Chara parpam was taken for the treatment of *Kamali*. Various studies have been carried out in this trial drug here. The study includes literary collections, Biochemical, Physico and Toxicological study Pharmacological study, and Clinical study. The drug has been selected for the treatment of *Kamalai* in reference with Pathartha guna vilakam Part II.Literary collections about the drug from various text books was done. It indicates the efficacy of the drug in the treatment of *Kamalai*. Chemical aspect deals with the identification, and ethno medicinal importance of the mineral. Gunapadam aspect expressed that the drug have protective property of Liver

Bio-chemical chemical analysis:

The analysis shows the precence of Sulphate, chloride, iron, calcium, pottasium. These elements are useful in many metabolic functions of our body.

Toxicological study:

Acute and Subaucute toxicological studies reveal that the drug *Chara parpam* does not having any toxic effect and safety of the drug is recorded through the histopathological results of animal model.

The haematological study results confirmed the safety of the drug. The liver and kidney function test of animal model in toxicological studies reveal the normal function and the vital organs and also registered the efficacy and safety of the drug.

Pharmacological study:

The results obtained from estimation of biochemical parameters suggesting that, Chara Parpam possess significant hepatoprotective property in CCL_4 induced liver toxicity in rat model.

Histopathology:

Histopathological studies of liver in toxicant group cause fatty changes, granular degeneration and inflammation. Also showed marked reduction in fatty degeneration and necrosis in animals treated with standard drug silymarin and Chara Parpam. It is evident that the Chara Parpam caused regeneration of liver parenchyma cells and treated hepatic cell damage due to CCL_4 toxicity.

Clinical study:

50 patients of both sexes were selected.

Among the 50 patients, 40 patients were treated as out- patients in the Post graduate department of Gunapadam, Govt.Siddha medical college hospital and Govt. Arignar Anna hospital, Arumbakkam, Chennai- 106. 10 patients were treated as in - patients.

The patients were observed regularly.

The clinical study was carried out with 50 patients. Three groups of cases were studied for the effect of *Chara parpam*. The patients had liver function examination before and after the treatment, including contents of serum proteins, total bilirubin (TB) and activities of ALT and AST.

The cumulative analysis revealed significant reduction in the levels of mean, SGOT, SGPT and ALP and Bilirubin. There was renormalization of protein levels. The normalcy of the above enzyme levels established the hepatoprotective effect of drug *Chara parpam* that might be able to induce accelerated regeneration of liver cell, reducing the leakage of the above mentioned enzymes into the blood.

In clinical study there is a marked improvement in signs and symptoms and Liver enzymes also. The results revealed that the drug possess 60% good relief, 18%atisfactory relief, 10% moderate relief mild relief, 12% cases there was no improvement

It shows the excellent safety and good efficacy of *Chara parpam* both Pre clinical and clinically. It has the potential to become an alternative to conventional therapy for Hepato cellular diseases.

6. CONCLUSION

Chara parpam is a distinctive type medicine, which is made up by a special type of preparation method mentioned in Pathartha guna vilakkam written by S.Kannusami pillai.

Literature survey, Physico chemical anatysis, Chemical analysis, was showed the efficacy of *charam* related to Liver diseases. Juice of *Justicia adathoda* is also having the traditional usage in treatement of liver diseases.

The finding of the pre clinical study suggests that effective role of *Chara parpam* on liver disease with fatty infiltration of liver with inflammation and hepatic necrosis to cirrhosis.

Chara parpam had remarkable effect on lipid with complete reduction in the lipid factors in the liver and its related enzymes. The metalloid drug could play similar role as extracellular calcium and it could evidence effective hepatoprotective drug.

Significant efficacy of the drug its human dose prescribed in the siddha literature evidences only therapeutic efficacy rather preventive role. It may be suggested as a drug to fatty liver which finds negligence cure in modern medicine.

This can be a definite alternative drug to modern allopathic drug in liver disease. The study reveals the possible mechanism of action on lipid lowering effect with potential anti oxidative role.

In clinical trials, the drug shows the significant improvement of 60% in Hepato cellular diseases.

Thus the innovative siddha science based *Chara parpam* had been proved by the modern scientific mechanism as a simple remedy to the complicated metabolic disorder in modern period.

7.SUMMARY

The herbo mineral preparation *Chara parpam* was prepared as per the classical way. This drug was subjected to various studies.

Chara parpam was selected for this study to establish the protection and efficacy of its Hepatoprotective activity on *Kamalai.*

I was collected the information about the drug, various text books, Literature was referred. From them, the author came to an idea about the drug and its efficacy on *Kalleral noi* a brief description about modern aspect and Siddha aspect of the mineral *Charam* and *Vediyuppu* its identifying characters and Physico – chemical data's were given.

The Physico – chemical analysis shows the precence of Sulphate, chloride, iron, calcium, pottasium. These elements are useful in many metabolic functions of our body.

Acute and Subacute toxicological studies show strong evidence of the non-toxic-effect of the *Chara parpam*. The results showed *Chara parpam* is safe and explained the extensive utilization of the Siddha medicine.

The pharmacological analysis showed that the drug has got significant Hepatoprotective Efficacy.

In clinical study, the drug has showed improvement in 60% of cases.

The patients were responding well from the beginning of the treatment and no adverse effects were reported.

This present study suggests that *Chara parpam* has remarkable medicinal value against the disease ***Kalleral noi***.

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Form: I

CONSENT FORM

I certify that I have disclosed all the details about the study in the terms readily understood by the patient.

DATE: **SIGNATURE**
NAME

CONSENT BY THE PATIENT

I have been informed to my satisfaction by the attending physician the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give reasons for doing so.

I ,exercising my free power of choice , hereby give my consent to be included as a subject in the clinical trial of **…….**for the treatment of**……..**

DATE: **SIGNATURE**
NAME

ஒப்புதல் படிவம்

ஆய்வுகுறித்தஅத்தனைதகவல்களையும் நோயாளிஎளிதில் புரிந்துகொள்ளும் வகையில் நோயாளிக்குவிளக்கியுள்ளேன் என்றுஉறுதியளிக்கிறேன்

தேதி: ஆய்வாளரின் கையொப்பம்:

பெயர்:

நோயாளியின் ஒப்புதல்

இந்தஆய்வுகுறித்த முழு தகவல்கள்,மருந்தின் தன்மை,எனதுஉடல் நலன் குறித்தஆய்வுகள்,ஆய்வுக்கானமருத்துவபரிசோதனைகள் மற்றும் சிகிச்சைவிபரங்கள் ஆகியஅனைத்தும் மருத்துவரால் முழுமையாகவிளக்கிக் கூறப்பட்டுள்ளது.

இந்தஆய்விலிருந்துஎந்தநிலையிலும்,எவ்விதகாரணமுமின்றிவிலகிக்கொள்ளஎனக்கு முழு சுதந்திரம் உள்ளதுஎன்பதையும் அறிந்திருக்கிறேன்.

இந்தஆய்வில்,……ஒருபயனாளி யாகஎன்னைஉட்படுத்திக் கொள்ளஏவ்விதமானநிர்பந்தமுமின்றிமுழுமனதுடன் சம்மதிக்கிறேன் என்பதைத் தெரிவித்துக் கொள்கிறேன்.

தேதி: கையொப்பம்:
பெயர்:

1. **DEPARTMENT OF POST GRATUATE GUNAPADAM BRANCH,**
GOVERNMENT SIDDHA MEDICAL COLLEGE,
CHENNAI - 106.
ARIGNAR ANNA GOVERNMENT HOSPITAL FOR
INDIAN MEDICINE AND HOMEOPATHY, ARUMBAKKAM,
CHENNAI - 106.
OPEN CLINICAL TRAIL PHASE II B
HAEMATINIC ACTIVITY ON *PULI ILAI CHOORANAM*,
FORM II

1. Centre :
2. Code no : Level of study: OPD/IPD
3. Name of the patient :
4. Address :

5. Age : : sex: Male ☐ Female ☐
6. Educational Status :
7. Occupation :
8. Income :
9. Religion : H ☐ M ☐ CH ☐ S ☐
10. Marital Status :

11. Date of Admission :
12. Date of Discharge :
13Diagnosis :

PERSONAL HISTORY:

11. Food habits : Veg ☐ Non Veg ☐ Veg/Egg ☐
12. Addiction : None ☐ Smoking ☐ Snuff ☐
Ganja ☐ Alcohol ☐ Opium ☐
13. Sleep : Good ☐ Distributed ☐ Insomnia ☐
14. Presence of anxiety : Yes ☐ No ☐
15. Naadi : Vatham ☐ Pitham ☐ Kapam ☐ Thontham ☐

FAMILY HISTORY:

1. Hypertension : Yes ☐ NO ☐
2. Diabetes mellitus : Yes ☐ NO ☐
3. Tuberculosis : Yes ☐ NO ☐
4. IHD/MI/MS/AS : Yes ☐ NO ☐
5. If any other disease specify : Yes ☐ NO ☐

PRESENTING SYMPTOMS:

		Yes	No	Duration (WEEKS)
1.	Pain :	☐	☐	_____
2.	Vomiting :	☐	☐	_____
3.	Indigestion :	☐	☐	_____
4.	Sore tongue :	☐	☐	_____
5.	Diarrhoea :	☐	☐	_____
6.	Constipation :	☐	☐	_____
7.	Haematemesis :	☐	☐	_____
8.	Loss of appetite :	☐	☐	_____
9.	Thirst :	☐	☐	_____
10.	Dysphasia :	☐	☐	_____
11.	Flatulence :	☐	☐	_____
12.	Heart-burn :	☐	☐	_____
13.	If any other disease :			_____

HISTORY OF PRESENT ILLNESS:

1. Pain :
2. Vomiting :
3. Indigestion :
4. Sore tongue :
5. Diarrhoea :
6. Constipation :
7. Haematemesis :
8. Loss of appetite :
9. Thirst :
10. Dysphasia :
11. Flatulence :
12. Heart-burn :
13. Bloating & abdominal fullness :
14. Water brash :
15. Nausea and copious vomiting:
16. Weight loss :
17. Melena :
18. If any other disease

PAST HISTORY:

1. Date of diagnosis : _____
2. Duration of disease : _____ months/years
3. Source of any infection : _____
4. Gastrointestinal bleeding : Yes ☐ No ☐ If Yes, when? _____
5. Cancer(GI tract) : Yes ☐ No ☐
6. Cancer(other organs) : Yes ☐ No ☐
7. Sedentary life style : Yes ☐ No ☐
8. Achlorhydria : Yes ☐ No ☐
9. Tumours in stomach : Yes ☐ No ☐
10. Tuberculosis (GI tract) : Yes ☐ No ☐
11. Diabetes : Yes ☐ No ☐
12. Nephritis : Yes ☐ No ☐
13. Syphilis : Yes ☐ No ☐
14. AIDS : Yes ☐ No ☐
15. Pt.underwent any surgery : Yes ☐ No ☐
16. UTI : Yes ☐ No ☐
17. Others : If Yes , Specify _____

HISTORY OF PREVIOUS TREATMENT: Yes ☐ No ☐

If yes, give details as follows:

PHYSICAL EXAMINATION:

1. Height (cm) : _____
2. Weight (cm) : _____
3. Pulse : _____
4. Blood pressure : _____
5. Temperature : _____
6. RR : _____ /minute
7. Anaemia : Present ☐ Absent ☐
8. Lymphadenopathy : Present ☐ Absent ☐
9. Pigmentation : Present ☐ Absent ☐

PRESENTING SIGNS:

1. Bloating and abdominal fullness : Present ☐ Absent ☐
2. Water brash (Rush of saliva after an episode of Regurgitation to Dilute the Acid in oesophagus) : Present ☐ Absent ☐
3. Nausea and copious vomiting : Present ☐ Absent ☐
4. Loss of appetite and weight loss : Present ☐ Absent ☐
5. Haematmesis : Present ☐ Absent ☐
6. Melena(tarry, foul-smelling faces

due to oxidized iron from Haemoglobin) : Present ☐ Absent ☐

7. Fatigue : Present ☐ Absent ☐
8. Heartburn : Present ☐ Absent ☐
9. Hunger : Present ☐ Absent ☐

CLINICAL EVALUATION:

1. Cardio vascular system : Normal ☐ Abnormal ☐ Details _____
2. Respiratory system : Normal ☐ Abnormal ☐ Details _____
3. Central nervous system : Normal ☐ Abnormal ☐ Details _____
4. Urogenital system : Normal ☐ Abnormal ☐ Details _____

SIDDHA PARAMETERS:

1. Naa : Normal ☐ Abnormal ☐ Details _____
2. Niram : Normal ☐ Abnormal ☐ Details _____
3. Mozhi : Normal ☐ Abnormal ☐ Details _____
4. Vizhi : Normal ☐ Abnormal ☐ Details _____
5. Malam : Normal ☐ Abnormal ☐ Details _____
6. Moothiram : Normal ☐ Abnormal ☐ Details _____
7. Sparisam : Normal ☐ Abnormal ☐ Details _____
8. Naadi : Normal ☐ Abnormal ☐ Details _____

RESULT:

1. GOOD : ☐
2. FAIR : ☐
3. POOR : ☐
4. NO RESPONSE : ☐

SIGNATURE OF MEDICAL OFFICER SIGNATURE OF INVESTIGATOR

LABORATORY INVESTIGATION AND CLINICAL PARAMETERS

1. Centre : _____
2. Code no : _____ Level of study: OPD/IPD
3. Name of the patient : _____
4. Age : : sex: Male ☐ Female ☐

5. Date and month of assessment : _____

CLINICAL PARAMETERS:

Urine: Albumin, Sugar, Deposits.

MOTION; Ova ,Cyst

HAEMOGRAM :Total WBC Count, RBC, Hb, ESR, Barium meal,Endoscopy,Ultra sound

2.

DEPARTMENT OF POST GRATUATE GUNAPADAM BRANCH,
GOVERNMENT SIDDHA MEDICAL COLLEGE,
CHENNAI-106.
ARIGNAR ANNA GOVERNMENT HOSPITAL FOR
INDIAN MEDICINE AND HOMEOPATHY, ARUMBAKKAM,
CHENNAI-106.
OPEN CLINICAL TRAIL PHASE II B
HEPATOPROTECTIVE ACTIVITY OF
"*CHARA PARPAM''*
FORM II

16. Centre :
17. Code no : Level of study: OPD/IPD
18. Name of the patient :
19. Address :

20. Age : : sex: Male ☐ Female ☐
21. Educational Status :
22. Occupation :
23. Income :

24. Religion : H ☐ M ☐ CH ☐ S ☐
25. Marital Status :

11. Date of Admission :
12. Date of Discharge :
13. Diagnosis :

PERSONAL HISTORY:

26. Food habits : Veg ☐ Non Veg ☐ Veg/Egg ☐
27. Addiction : None ☐ Smoking ☐ Snuff ☐
Ganja ☐ Alcohol ☐ Opium ☐
28. Sleep : Good ☐ Distributed ☐ Insomnia ☐
29. Presence of anxiety : Yes ☐ No ☐
30. Naadi : Vatham ☐ Pitham ☐ Kapam ☐ honthan ☐

FAMILY HISTORY:

6. Hypertension : Yes ☐ NO ☐
7. Diabetes mellitus : Yes ☐ NO ☐
8. Tuberculosis : Yes ☐ NO ☐
9. IHD/MI/MS/AS : Yes ☐ NO ☐
10. If any other disease specify : Yes ☐ NO ☐

PRESENTING SYMPTOMS:

	Yes	No	Duration (WEEKS)
14. Anorexia :	☐	☐	_____
15. Nausea :	☐	☐	_____
16. Vomiting :	☐	☐	_____
17. Fatigue :	☐	☐	_____
18. Insomnia :	☐	☐	_____
19. Yellow colored eyes :	☐	☐	_____
20. Yellow colored urine :	☐	☐	_____
21. Pale coloured motion :	☐	☐	_____
22. Skin itching :	☐	☐	_____
23. Pain abdomen :	☐	☐	_____
24. Fever :	☐	☐	_____
25. Diarrhoea :	☐	☐	_____
26. Hematemesis :	☐	☐	_____
27. If any other disease :	_____		

HISTORY OF PRESENT ILLNESS:

Onset of Jaundice : Acute ☐ Chronic ☐

PAST HISTORY:

18. Date of diagnosis : _____
19. Duration of disease : _____ months/years
20. Source of HBV infection : _____
21. High risk Group : Medical worker Paramedical worker
Sex worker others
Non – High risk group
22. Alcohol : Yes ☐ No ☐
23. Cancer(other organs) : Yes ☐ No ☐
24. Sedentary life style : Yes ☐ No ☐
25. Achlorhydria : Yes ☐ No ☐
26. Tuberculosis : Yes ☐ No ☐
27. Diabetes : Yes ☐ No ☐
28. Nephritis : Yes ☐ No ☐
29. Syphilis : Yes ☐ No ☐
30. AIDS : Yes ☐ No ☐
31. Pt.underwent any surgery : Yes ☐ No ☐
32. UTI : Yes ☐ No ☐
33. Others : If Yes , Specify _____

HISTORY OF PREVIOUS TREATMENT: Yes No ☐ ☐
If yes, give details as follows:

PHYSICAL EXAMINATION:

10. Height (cm) : _____
11. Weight (cm) : _____
12. Pulse : _____
13. Blood pressure : _____
14. Temperature : _____
15. RR : _____ /minute
16. Anaemia : Present ☐ Absent ☐
17. Lymphadenopathy : Present ☐ Absent ☐
18. Pigmentation : Present ☐ Absent ☐

PRESENTING SIGNS:

10. Odema feet : Present ☐ Absent ☐
11. Icterus : Presen ☐ Absent ☐
12. Spider : Present ☐ Absent ☐
13. Gynacomastia : Present ☐ Absent ☐
14. Liver :Palpable ☐ Not palpable ☐
15. Splenomegaly : Present ☐ Absent ☐
16. Ascites : Present ☐ Absent ☐
17. Encephalopathy : Present ☐ Absent ☐

CLINICAL EVALUATION:

5. Cardio vascular system : Normal ☐ Abnormal ☐ Details _____
6. Respiratory system : Normal ☐ Abnormal ☐ Details _____
7. Central nervous system : Normal ☐ Abnormal ☐ Details _____
8. Urogenital system : Normal ☐ Abnormal ☐ Details _____

RESULT:

5. GOOD : ☐
6. FAIR : ☐
7. POOR : ☐
8. NO RESPONSE : ☐

SIGNATURE OF MEDICAL OFFICER SIGNATURE OF INVESTIGATOR

LABORATORY INVESTIGATION AND CLINICAL PARAMETERS

5. Centre : _____
6. Code no : _____
   Level of study : OPD/IPD
7. Name of the patient : _____
8. Age : : sex: Male ☐ Female ☐

5. Date and month of assessment : _____

CLINICAL PARAMETERS:

Urine: Albumin, Sugar, Deposits,Bile pigments, Bile Salts

Urine: Albumin, Sugar, Deposits.
MOTION; Ova ,Cyst
HAEMOGRAM :Total WBC Count, RBC, Hb, ESR,
Liver function test:
AST,ALT,ALP, Bilirubine, total protein, Albumin., Prothrombin time

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to Dr/~~Mr/Mrs~~ P. ARUNMOZHI B.S.M.S

for participating in the Workshop on

'Introduction to Scientific & Medical Writing'

organized by the Department of Epidemiology,
The Tamil Nadu Dr. M.G.R. Medical University on 18th March, 2011.

This educational activity has been awarded **10 Credit Points**
by the Centre for Accreditation, The Tamilnadu Dr. M.G.R. Medical University.

Dr. N. KABILAN, M.D. (Siddha)
HOD i/c, DEPT. OF EPIDEMIOLOGY

Dr. SUDHA SESHAYYAN, M.S.
REGISTRAR (FAC)

Dr. MAYIL VAHANAN NATARAJAN
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. D.Sc. F.R.C.S. D.Sc. (Hon)[3]
VICE CHANCELLOR

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

69, Anna Salai, Guindy, Chennai - 600 032.

DEPARTMENT OF SIDDHA

CERTIFICATE OF PARTICIPATION

This is to certify that Dr/Mr/Ms P. Arunmozhi *has participated in the CME on* Good Clinical Practice *conducted by Department of Siddha on* 25-01-2011.

This educational activity has been awarded 2 *Credit points by The Centre for Accreditation, The Tamil Nadu Dr. MGR Medical University.*

Total Credits Claimed :

Participant's Signature Date

Dr. N. KABILAN
Prof & Head
Department of Siddha

Dr. SUDHA SESHAYYAN
Registrar i/c

Dr. MAYIL VAHANAN NATARAJAN
Vice Chancellor

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to Dr. P. ARUNMOZHI

for participating as a ~~Resource Person~~ / Delegate in the V Workshop on

"Research Methodology & Biostatistics"

for AYUSH Post-Graduates & Researchers

organized by the Department of Siddha,

The Tamil Nadu Dr. M.G.R. Medical University

from 8th August 2011 to 12th August 2011.

Dr. MAYILVAHANAN NATARAJAN
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. D.Sc. F.R.C.S. D.Sc. (Hon)^3
VICE CHANCELLOR

Dr. SUDHA SESHAYYAN, M.S.
REGISTRAR (FAC)

Dr. N. KABILAN, M.D. (Siddha)
HOD, DEPT. OF SIDDHA

VEL'S COLLEGE OF PHARMACY

Approved by the Government of Tamil Nadu
Affiliated to The Tamil Nadu Dr. MGR Medical University
Velan Nagar, P.V. Vaithiyalingam Road, Pallavaram, Chennai - 600 117
Phone : (91-44) 2266 2500 / 01 / 02 / 03 Fax : (91-44) 2266 2513
E-mail : velscollege@gmail.com Web : www.velscollege.com

S.No	Title of The Project	Name of The Investigator	Approval status/Remarks	Project Reference
39.	Preclinical study on "Singa Chooranam" for Bronchodilator Activity in the management Eraippu..	Dr. T. Sathya	Totally 48rats were proposed and sanctioned. Altered method was suggested. it was advised to share the control and standard group results	XIII/VELS/PCOL/ 39/2000/CPCSEA/I AEC/08.08.12
40.	Preclinical study on "Eraip Mathirai" for Bronchodilator Activity in the management Eraippu.	Dr. S. Savitha	Totally 48rats were proposed and sanctioned. Altered method was suggested.	XIII/VELS/PCOL/ 40/2000/CPCSEA/I AEC/08.08.12
41.	Preclinical study on "Kuzhpaar Chooranam" for Styptic Activity in the management perumbadu.	Dr. S. Savitha	Total number of animals proposed were 40mice. Permitted to proceed. But it is advised to share the common group data with similar pattern of projects if possible.	XIII/VELS/PCOL/ 41/2000/CPCSEA/I AEC/08.08.12
42.	Preclinical study on "Sarakon Poo Chooranam" for Hepatoprotective Activity in management of Kamalai.	Dr. R. Venkatesh	40rats were proposed and Sanctioned	XIII/VELS/PCOL/ 42/2000/CPCSEA/I AEC/08.08.12
43.	Preclinical study on "Kanc Chenduram" for Hypoglycemic Activity in the management Madhumegam (Diabetes mellitus	Dr. R. Venkatesh	40rats were proposed and Sanctioned	XIII/VELS/PCOL/ 43/2000/CPCSEA/I AEC/08.08.12
44.	Haematinic activity of Tamirinc indicus	Dr. Arun Mozhi	Total number of animals proposed was 42 rats. But 36 rats were Sanctioned.	XIII/VELS/PCOL/ 44/2000/CPCSEA/I AEC/08.08.12

City Centre : No. 521/2, Anna Salai, (Opp. G.R. Complex), Nandanam, Chennai - 600 035.
Phone / Fax : (91-44) 2431 5541 / 2431 5542 **E-mail :** velsrinivasa@vsnl.net

Dr. J.ANBU, M.Pharm., Ph.D., D.M.L.T.,MBA.,
Professor & Head
Department of Pharmacology & Toxicology
School of Pharmaceutical Sciences
Vels University
Pallavaram, Chennai-600 117,

VEL'S COLLEGE OF PHARMACY

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Affiliated to The Tamil Nadu Dr. MGR Medical University
Velan Nagar, P.V. Vaithiyalingam Road, Pallavaram, Chennai - 600 117
Phone : (91-44) 2266 2500 / 01 / 02 / 03 Fax : (91-44) 2266 2513
E-mail : velscollege@gmail.com Web site : www.velscollege.com

XIII INSTITUTIONAL ANIMAL ETHICS COMMITTEE MEETING

Date: 08.08.2012 and 11.08.2012 **Time**: 2.30 P.M.

Venue: Conference Hall, Vels College Pharmacy

XIII Institutional Animal Ethics Committee meeting was held as per the norms of CPCSEA and the enclosed list of members attended the meeting and discussed various project proposals submitted by the investigators. The details of the meetings and approval status of the proposed project are as follows

S.No	Title of The Project	Name of The Investigator	Approval status/Remarks	Project Reference
1.	Hepatoprotective activity of Charaparpam by CCL4 induced method in rats	Dr. Arun Mozhi	Total number of animals proposed was 42 rats and after having discussion it was decided to reduce 12 number of animals and suggested to share the standard group results with other researchers who has planned to carryout similar kind of study. And also to follow OECD 425 method for acute toxicity study.	XIII/VELS/PCOL/ 01/2000/CPCSEA/I AEC/11.08.2012
2.	A study on Poovarampattai kudineer choornam for the treatment of Swethakuttam.	Dr. G. Kala	Total number of animals proposed was 42 mice. But 20rats were Sanctioned.	XIII/VELS/PCOL/ 02/2000/CPCSEA/I AEC/11.08.2012
3.	Evaluation the therapeutic efficacy of Soothagathaiudaikkum kasayam in soothagavayu.	Dr. K .Dhanalakshmi	Total number of animals proposed was 40 rats, and it was advised to minimize the number to 30 rats only and suggested to reuse the animals sanctioned for safety study after recovery.	XIII/VELS/PCOL/ 03/2000/CPCSEA/I AEC/11.08.2012

City Centre : No. 521/2, Anna Salai, (Opp. G.R. Complex), Nandanam, Chennai - 600 035.
Phone / Fax : (91-44) 2431 5541 / 2431 5542 **E-mail :** velsrinivasa@vsnl.net

Dr. J.ANBU, M.Pharm., Ph.D., D.M.L.T., MBA.
Professor & Head
Department of Pharmacology & Toxicology
School of Pharmaceutical Sciences
Vels University
Pallavaram, Chennai-600 117.

PARC PLANT ANATOMY RESEARCH CENTRE

Herbal PARC

Dr.P. Jayaraman, Ph.D.
Director, PARC,
Retd. Professor, Presidency College

AUTHENTICATION CERTIFICATE

Based upon the ~~Organoleptic~~ /macroscopic /~~microscopic~~ examination of fresh /~~market~~ sample, it is certified that the specimen given by Dr.P.ARUNMOZHI, P.G. GUNAPADAM, Govt. Siddha Medical College, Chennai is identified as below:

Binomial: *Tamarindus indica L.*

Family: Caesalpiniaceae

Synonym(s): —

Regional names: Tam. Puli

Reg.No of the certificate: PARC/2012/1471.

References: Nair, N.C & Henry, A.N. Flora of TamilNadu, India I: pg:133. .1983.

Henry, A.N. *et al.* Ibid. II: — .1987.

Ibid. III: — .1989.

Date: 04/03/12.

(Prof.P.JAYARAMAN)
Prof.P.Jayaraman,Ph.D.
Director,
Institute of Herbal Botany
PLANT ANATOMY RESEARCH CENTRE,
No.4-II Street,Sakthi Nagar,
West Tambaram,Chennai-45.
Ph:044-22263236, Cell:8939136959
E-mail:herbalparc@yahoo.com

#4,2nd Street, Sakthi Nagar,
West Tambaram, Chennai-600 045
Ph:044-22263236,+918939136959
Email- herbalparc@yahoo.com

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

69, Anna Salai, Guindy, Chennai - 600 032.

DEPARTMENT OF SIDDHA

CERTIFICATE OF PARTICIPATION

This is to certify that Dr/Mr/Ms P. Arunmozhi *has participated in the CME on* Pharmacological and Toxicological Studies *conducted by Department of Siddha on* 29-11-2010.

This educational activity has been awarded 2 *Credit points by The Centre for Accreditation, The Tamil Nadu Dr. MGR Medical University.*

Total Credits Claimed :

Participant's Signature Date

Dr. N. KABILAN
Prof & Head
Department of Siddha

Dr. SUDHA SESHAYYAN
Registrar i/c

Dr. MAYIL VAHANAN NATARAJAN
Vice Chancellor